MAY 24 1926

Medical Lib.

Vol. XXXVI MAY, 1926 No. 5

THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
DEVOTED TO DISEASES OF THE

EAR - NOSE - THROAT

FOUNDED IN 1896 BY

DR. M. A. GOLDSTEIN.

Managing Editor and Publisher.

COLLABORATORS.

Dr. ALBERT A. GRAY, Glasgow;
Dr. THOMAS GUTHRIE, Liverpool;
Dr. CHEVALIER JACKSON, Phila.;
Dr. F. LASAGNA, Parma;
Dr. M. D. LEDERMAN, New York;
Dr. HARRIS PEYTON MOSHER, Boston;
Dr. FRANCIS R. PACKARD, Phila.;
Dr. W. SCHEPPEGREL, New Orleans;
Sir ST. CLAIR THOMSON, London;
Dr. W. A. WELLS, Washington, D. C.;
Dr. D. J. GIBB WISHART, Toronto;
Dr. SIDNEY YANKAUER, New York;

For Contents See Page 1.

Subscription, $6.00 per Annum, in Advance.

Foreign Subscription, 35 Shillings per Annum, Post Free.

Single Copies, 75 cents.

PUBLISHED BY THE LARYNGOSCOPE CO.

3858 Westminster Place, St. Louis, Mo., U. S. A.

FOREIGN OFFICE, BAILLIERE, TINDALL & COX,

8 HENRIETTA ST., STRAND, LONDON, ENG.

[Entered at the Postoffice at St. Louis, Mo., as Second Class Matter, in July, 1896.]

THE

LARYNGOSCOPE.

VOL. XXXVI MAY, 1926 No. 5

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE PATHOLOGY OF DEAF-MUTISM.*

DR. F. R. NAGER, Linck, Switzerland.

Before entering upon the subject of my lecture, I wish to express my cordial thanks for the honor of your invitation to take part in your annual meeting as your guest. I am fully conscious of the distinction conferred, but also of the duties connected with my task and trust that I shall fulfill your expectations.

I should, however, like to bestow the greater part of this honor upon my two teachers, the deceased Professor Bezold, of Munich —whose remarkable textbook has been translated excellently in Chicago by our colleague, Dr. Hollinger—and upon Professor Siebenmann in Bale, whom many of you will remember personally from his visit to Boston in 1912. With heartfelt gratitude I would recall to your memory the great merits of this prominent Swiss otologist.

My scientific pedigree prompted me to choose the subject of deaf-mutism for this lecture, because through their research work Bezold greatly advanced the clinical and Siebenmann the pathological aspect of this subject.

The pathology of deaf-mutism, about which I should like to give you a general survey, is a new field of work in otohistology, which could only be entered upon successfully after the introduction of the Celloidin-technique.

The macroscopic changes in acquired deaf-mutism were well known to the older investigators, but the minute alterations in the labyrinth of congenital deaf-mutism could only be ascertained by the modern microscopic technique.

*Presented before the American Academy of Ophthalmology and Oto-Laryngology, Chicago, Oct. 20, 1925.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Oct. 26, 1925.

In the first decade of our century, when this research work became known, it was supposed that certain alterations were characteristic of deaf-mutism, just because similar changes were found in the labyrinth of congenitally deaf animals, for example Japanese dancing mice, albino cats and dogs, etc.

Later on, however, it was shown, that analogous abnormalities may exist also in cases of simple deafness. Furthermore, similar anomalies were described in experimental labyrinth pathology and to this discovery we owe much insight into the pathogenesis of inner ear diseases.

It may be added, that in isolated cases histological research did not result in finding any local changes. It is supposed, therefore, that there must exist anomalies of the *central* connections and brain. According to present requirements examination of the inner ear should be followed by an examination of the central connections. And indeed the neurologists repeatedly ascertained changes in these structures, though it remains for future examinations to demonstrate which alterations are primary or secondary and which are the cause of the defect in hearing.

The "pathology of deaf-mutism" for this reason does not represent something that is entirely confined to the labyrinth of the deaf-mute. A résumé under this designation offers, nevertheless, so much that is instructive and interesting, especially when experiences gained in the other researches of otohistology are also utilized, that even future otologists will continue to cultivate this interesting field.

In the same way the conception of deaf-mutism, though accepted generally as a clinical term, scientifically is not quite correct, because according to Bezold's hearing tests in fact only a small number of so-called deaf-mutes are *totally* deaf; the latter belong mostly to the acquired forms of deafness. Quite a large number of the so-called congenital cases on the other hand exhibit very considerable remains of hearing available for aural education.

There exists therefore no distinct line of demarcation between deaf-mutism and *simple* deafness, not only in their pathological, but also in their *clinical* aspect. It is not the high degree of deafness alone, but its existence in early childhood, when speech should be acquired with the aid of hearing, that leads to deaf-mutism.

I should like to disregard in this lecture the *central disturbances* and their clinical effects on the faculty of hearing and the acquirement of speech, and to limit myself entirely to deaf-mutism and its local manifestations in the ear. But I should like to emphasize, nevertheless, that future deaf-mute examinations will have to in-

clude still more thoroughly the central disturbances and their histological changes.

For a long time, a distinction was made *clinically* between congenital and acquired deaf-mutism, which proved to be very useful, but which is not scientifically exact on closer scrutiny. We depend here predominantly on the statements of the child's parents which are often unreliable. In addition a certain number of parents very unwillingly admit, even if they know it, the congenital nature of a defect.

Moreover, it is not always possible to decide even by clinical examinations whether deaf-mutism be congenital or acquired; there will always remain uncertain cases.

The hope of finding criteria in the *microscopical* investigations, which would permit of definite distinction between these two forms, also proved to be deceptive because similar histological changes could be found in both forms.

With increasing knowledge of the histological abnormalities the need arose to group them in some manner. This, however, met with considerable difficulties, especially as to the classification of *congenital* deaf-mutism. According to our knowledge today, there is as yet no method of classification, which satisfies *all* demands, and at the *same time* embodies a proper consideration for clinical, etiological, histological and pathogenic points of view.

We will, therefore, have to group the cases, as we become interested in special questions, in such a way that we may understand how analogous *pathological* changes may be found, under quite different *etiological factors*. I shall now submit to you a certain classification in accordance with the work of Siebenmann, taking into consideration the results of late researches.

Acquired deaf-mutism is today explored most thoroughly, because the results of the general and experimental labyrinth histology have substantially assisted in clearing up this field. We deal in most of these cases with the results of *inflammatory* processes in the *labyrinth*. Their origin may be meningitic, tympanic or hematogenic (metastatic). Noninflammatory affections, as for instance, injuries of the skull, are the cause of labyrinth disease in the *minority* of cases.

As far as *labyrinthitis* is concerned, we know today all its grades and different stages up to a state of healing, so that we are able to interpret the alterations found in deaf-mutism arising from this cause. The meningitic cases are most frequent because a number of diseases to be considered in this connection may occur as epidemics. (Meningitiscerebrospinalis, scarlet fever, measles, parotitis, typhoid).

Deaf-mutism caused by meningitis has been known longest and best. Each epidemic was always followed by an increase of deafness, resp. of deaf-mutism. In such cases we find a new formation of bone and connective tissue of different degrees up to the complete obliteration of the labyrinth cavities. If the cochlear duct is preserved, it will show important changes, such as collapses, dilatations or adhesions in its interior; Corti's organ is missing, so are the cells of the stria vascularis. The membrana tectoria is often enclosed in an epithelial capsule. The spiral ganglion and nerve fibres are absent. In isolated cases a fistula into the middle ear could also be traced.

That *scarlet fever* and *measles* can likewise lead to *meningitic* labyrinthitis had to be assumed from the results of *clinical* examinations of deaf-mutes with normal drumheads. I shall later on show you some slides of a case of deaf-mutism following scarlet-meningitis, showing an almost complete bony obliteration of the labyrinth.

In cases of *measles,* especially bronchopneumonic complications are to be feared and this may be followed by metapneumonic *meningitis* with consecutive deaf-mutism.

Moreover, deaf-mutism after parotitis and hereditary syphilis seems to be due to meningitic labyrinthitis, according to the clinical and the few microscopical investigations.

Tympanic labyrinthitis also occurs frequently and has become well known as a cause of acquired deaf-mutism. Every necrotic otitis media endangers the inner ear through the possibility of an invasion of the labyrinth. All of us are familiar with such cases of deafness in childhood, followed by deaf-mutism after scarlet fever, measles, influenza-otitis as well as after tubercular and congenital syphilitic middle ear suppuration.

Histological examinations disclose very extensive destructions even up to an obliteration of the labyrinth, not infrequently coupled with cholesteatoma. Aside from the new formations of bone and connective tissue, the route of labyrinthine infection may be found as a healed fistula. Not infrequently the remains of the membranous labyrinth can be traced, the changes being similar to those found in cases of meningitic labyrinthitis.

A *metastatic* labyrinthitis has to be accepted as the cause of *deaf-mutism* after osteomyelitis, but as yet this view has not been confirmed by histological investigations.

In cases of *noninflammatory* labyrinthic affections, injuries to the skull in earliest childhood assume first place as a cause of deaf-mutism. Here also labyrinth pathology has greatly clarified our

knowledge. A manifestly greater importance has of late been attached to injuries during birth, since the labyrinths of children, who have died *intra* and *post partum* may show changes, which correspond entirely to those found after severe injuries of the skull.

The labyrinths of such deaf-mutes are partly filled with bony and connective tissue; the scala media in a case observed by us showed tremendous dilatation; the spiral ganglion and nerve fibres had been destroyed almost entirely. The conditions correspond to the after-effects of *inflammatory* labyrinthitis.

From the *clinical* investigation of deaf-mutes and from the histological examinations of persons who became deaf, we have to reckon also with the possibility that isolated affections of the *auditory nerve,* as well as of Corti's organ may lead to acquired deaf-mutism as the result of a neuritis with consecutive atrophic degeneration. Such postinfectious or toxic neuritis not infrequently appears after the diseases, heretofore referred to (meningitis, scarlet fever, measles, diphtheria, parotitis, typhoid). Further a toxic neuritis of the cochlear nerve would explain the presence of a normal drumhead with normal reaction of the static labyrinth in these deaf-mutes.

Exact histological investigations of such well defined cases have not as yet been described, but findings of that character may possibly have been included in reports on certain cases of congenital deaf-mutism.

The pathology of *congenital* deaf-mutism is much more many-sided.

First of all I would like to comment upon *endemic congenital deaf-mutism,* since the clinical, pathological and etiological aspect is well known in Switzerland. Here we are dealing with one of the symptoms of *endemiccretinic degeneration,* not uncommon in certain mountain regions (*i. e.,* in the Alps of central Europe and in central Asia) having similar hygienic, economic, geologic and climatic conditions.

The central nerve system, the thyroid gland, the skeleton and the ear being affected in different degrees, afford us a varied picture of endemic cretinism, which in its greatest development, is represented by the idiotic, goiterous, deaf-mute dwarf.

The pathological changes in endemic congenital deaf-mutism affect in a very peculiar way the medial tympanic wall. There is a marked hyperostosis of the promontory, with a narrowing of the window niches and adhesions of the deformed stapes.

In the cochlea, a small hyaline body which is embedded between Corti's organ and the membrana tectoria, has been hitherto found only in endemic deaf-mutes.

Except for this, the spiral organ of Corti and the nerve fibres may be normal, as such patients generally have considerable remains of hearing.

These striking changes in the ear are probably due to noxious influences in human development, during the last third of the foetal period. Neurologists also have reached similar conclusions based on the results of histological investigations of cretinic brains.

These changes are so typical, that the diagnosis of the *endemic* form becomes possible also post-mortem and without clinical data. In cretinic dogs similar abnormalities have also been ascertained. It may be mentioned incidentally that in Switzerland the most severe forms of the endemic cretinic degeneration are steadily decreasing, due to persistent improvements of hygienic conditions, the opening up of isolated districts, the fight against endemic goiter with prophylactic iodin therapy.

In sporadic deaf-mutism the changes are exclusively in the labyrinth, mostly in both ears. They have to be looked upon as malformations, though it is not certain as yet to what extent they are to be considered as congenital defects or as consequences of intrauterine inflammations.

The alterations may exhibit different *grades* of development, which have led to a division into different groups and types.

The complete absence of the labyrinth and pyramid is very rare (Group A). In the more frequent malformations of the *auricle* (microtic and atresia auris congenital) the inner ear is less affected or not all.

Much more numerous on the other hand, are the slighter changes in the labyrinth, which are either confined to the neuroepithelium of the scala media of the cochlea (Group B), or may be associated with anomalies of the sacculus as well as of the cochlear duct (Group C). The opinions about the congenital character of the changes in Group B have begun to waver lately. Early postfetal degenerative atrophic processes in the eighth nerve and its neuroepithelium, perhaps also birth injuries might play here a causal role.

The third group (C) with sacculocochlear degenerations contains the greater part (70 per cent) of sporadic congenital deaf-mutes with remnants of hearing. It includes most of the constitutional hereditary form. Here also different grades of development are observed: those with intact modiolus, those with defective central axis and finally those with large dilations of the saccus endolymphaticus (type Mondini). The last can easily be observed macroscopically and has already attracted the attention of older authors. Here even the bony labyrinth is much affected. The neuroepithelium, the spiral

organ and the nerve fibres likewise show extensive degenerations. In the cochlear duct there are folds and adhesions between Corti's organ and stria vascularis. The utricule and the semicircular canals are normal, so are their functions.

Certain similarities to the changes in cases of meningitic and experimental labyrinthitis caused Siebenmann to assume intrauterine inflammatory conditions in the labyrinth or in the cranial fossae as the cause of a number of these alterations.

Besides these forms, very marked malformations of the cochlea may exist, as for instance the presence of only 1½ coils, instead of the normal 2½ coils.

Quite analogous results were obtained from the histological examination of congenitally hard of hearing patients. It is very interesting to observe that tuning fork examination revealed defects even in the inferior tone limit in cases in which the upper coils of the cochlear were absent.

It may be added that in quite a considerable number of these cases the bony labyrinths contains distinct spots of *otosclerosis*. This is probably not due to accident, as we know that on the other hand the *cochlea* of *otosclerotics* very often shows slight malformations.

It must be left to future investigations with the aid of more observations and a faultless technique to shed an increased light, especially on etiology and pathology.

To effect this, we shall have to take into consideration the results of the general labyrinth pathology, secondly of other ear and brain malformations and, lastly, of experimental and comparative anatomical studies in general.

I have attempted to outline a survey of the pathology of deaf-mutism. It is still a mosaic, like ancient pictures, showing many a gap, rendering a general impression very difficult. But if we all, not only we in the old, but also you in the new world, with its magnificently equipped laboratories, will continue to work ahead, then a more perfect picture in all its details will be unveiled.

This knowledge surely is not only of scientific but also of practical interest, because from it therapeutic and prophylactic viewpoints will arise, which probably will reduce the frequency of deaf-mutism.

I wish to express the hope that this short lecture has awakened in you the interest and the desire for mutual work, through which the high aim which you seek to effect by an *international* exchange of thoughts and experiences may be materially advanced.

ACUTE MASTOIDITIS WITHOUT APPARENT MIDDLE EAR SYMPTOMS.*

DR. MORRIS A. WEINSTEIN, Philadelphia.

Mastoiditis often presents unusual difficulty in diagnosis. It is especially true in cases where the middle ear symptoms are negligible; and errors in diagnosis of this condition occur much more frequently than may be generally supposed. There are cases reported in both general and special literature, but under divers captions—such as "Atypical Mastoiditis," "Mastoiditis Without Aural Discharge," and "Primary Mastoiditis."

It would therefore be of great advantage to adopt for these cases one general name, which would avoid confusion.

Dabney,[1] in 1915, reported twenty-four cases from the literature, to which he adds two of his own and refers to them as "Idiopathic Mastoid Abscess." By this he means an abscess in the mastoid process without any immediately preceding or accompanying inflammatory involvement of the tympanic cavity.

He explains the origin and progress of these abscesses in the following manner. The infection starts, as usual, in the oropharynx, travels through the tube into the tympanic cavity, without finding conditions favorable for growth, and then passes on through the aditus into the mesh of mastoid cells. Here, the blood supply being poor and the cavity practically a closed one, the conditions found are ideal for a suppurative activity. The muco-periosteal lining of the antrum swells and the contiguous mucosa of the aditus follows suit, thus practically closing the narrow opening and completely walling off the antrum and mastoid cells from the tympanic cavity.

Salinger,[2] in 1920, speaking about primary subperiosteal abscess of the mastoid, which may or may not extend to the cortex, stresses the point that the cases may properly be termed primary only when they relate to a local trauma or a systemic infection.

Levi[3] quotes Zaufal, of Prague, who demonstrated, post-mortem, that the mastoid cells can undergo necrotic inflammatory changes without the middle ear being involved.

Ersner,[4] in 1922, reported seven cases of "Atypical Mastoiditis."

Hempstead,[5] in 1923, reviewed the literature and reported twenty-six cases since Dabney's report in 1915. He calls attention to the

*Read before the Philadelphia Laryngological Society, Jan. 5, 1926.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Jan. 13, 1926.

fact that in some reports the descriptions are meager, while a number of the others are mere cases rather of "Latent Suppurative Otitis Media." He further writes that mastoiditis without involvement of the middle ear should not be confused with latent suppurative otitis media, for in the latter there is deafness, sometimes pain, but no spontaneous discharge of pus. The drum membrane is without lustre, full, and sometimes bulging, and paracentesis is always followed by discharge of pus; mastoid involvement and other complications may attend; whereas, in the cases of mastoiditis without involvement of the middle ear, paracentesis is never followed by the discharge of pus.

In the cases of "Latent Suppurative Otitis Media" mentioned by Hempstead, I believe the causative germ in a majority of cases to be the "Streptococcus Mucosus." In the cases decisively traced to this germ, similar subjective and objective symptoms are encountered; lustreless drum, slight discharge after paracentesis, and a consequent pathology and bone necrosis far out of proportion to that observed in any other kind of infection.

King,[6] in 1923, reported a case of acute mastoiditis without aural discharge, in a woman, age 19 years; onset five weeks prior with a bilateral tonsillitis, two days later earache in the left ear, followed by pain and swelling over the left mastoid, drumhead dull red, no light reflex, no bulging, and no perforation.

Amsden,[7] in 1924, reported an unusual case of mastoiditis which had been operated on four years previously and at that time no pathology was found. After the operation the patient at intervals complained of pain in the ear, and four years later had an acute suppurative otitis media with spontaneous perforation. The tissues over the mastoid were thickened, reddened, and tender. Mastoidectomy was again performed and polypoid degeneration of the mucosa lining the mastoid cells was found.

Heggie and Knauer[8] reported two cases of mastoiditis without apparent involvement of the Middle ear. One was in a three-year-old girl, who complained of pain in the right ear with swelling behind the auricle; the nose, nasopharynx, larynx and both tympanic membranes were normal. The operation revealed an advanced mastoiditis. In their other case, almost the same symptomatology was present. The drum was incised but no pus or serum obtained. Operation disclosed a perforation near the floor of the antrum and cell necrosis.

Pugnat,[9] in 1924, reported a case of "recurring mastoiditis" four years after a previous suppurative otitis media, and mastoidectomy was followed by complete recovery. The mastoid was again involved,

after the patient developed an infection in the right thumb, and the pus in both mastoid and thumb showed the same infective organism —staphylococcus aureus. The writer believes this a case of mastoiditis of hematogenous origin.

Beeger,[10] in 1924, reported eighteen cases in whom the middle ear symptoms were almost negligible, whereas the mastoid symptoms were so dominant as to suggest primary mastoiditis. The author assumes a special strain of streptococcus against which the tympanic mucosa is immune allowing the infection to travel through into the antrum and blocking off the latter from the tympanic cavity.

Wagers,[11] in June, 1924, reported a case of acute mastoiditis apparently primary in an infant of seven months. The previous medical history in this case shows the patient had been fretful with movements of the hand towards the diseased ear, even before the appearance of postauricular swelling. This case might better be termed acute mastoiditis without apparent middle ear symptoms.

Diehl,[12] in July, 1924, reported a case of mastoiditis-epidural abscess without apparent middle ear involvement. The condition started in April, 1923, with rhinitis, stuffiness, and fullness in the right ear, without pain. The drums were slightly retracted and congested. In July the patient complained of severe pain behind the ear, due to swelling and tenderness posterior to the external canal. The canal and the drum were negative. Operation revealed a complete destruction of the mastoid cells. The mastoid cavity and antrum were filled with broken material. The cortex posterior to the mastoid was necrosed and pus was found between the cortex and the dura. The drum was incised at the close of the operation. No pus was found nor did it appear subsequently.

Brown,[13] in August, 1924, reported a case of atypical mastoiditis which was operated on two months after the initial onset of pain in the right ear. There was no evidence of tympanic membrane involvement at the time of operation, which disclosed extensive bone destruction of the mastoid, cells around the sinus, and the horizontal canal. Bone scrapings showed streptococcus in pure culture.

Gleason[14] in his Manual of Diseases of the Nose, Throat and Ear refers to the atypical cases as those where the disease progresses without well marked symptoms—the so-called "latent mastoiditis."

Kopetzky and Almour,[15] in THE LARYNGOSCOPE, Oct., 1925, reported three cases of primary acute mastoiditis. In their comment they lay stress on the blocking of the infection in the antrum, by granulations, away from the tympanic cavity.

Report of Case: History: R. B., female infant, age 14 months, was first seen by me, Apr. 21, 1924, with a history of restlessness

for the past week with no fever; nor were other disquieting symptoms noticeable. That evening, while giving her baby the usual bath, before putting her to bed, the mother by mere chance noticed a swelling behind the left ear. On most careful inquiry, the mother was absolutely positive in her statement that at no time was there any discharge from the ear, nor any other symptom to arouse a suspicion of ear trouble.

Family history: negative.

Previous medical history: no previous illness.

Examination: The examination reveals a postauricular swelling, pushing the auricle forward, the skin covering was dusky red, and some fluctuation was elicited on palpation. Both drums, including the nose and nasopharynx, show no pathological changes.

I incised the left eardrum but no pus could be obtained nor was there any discharge from this ear subsequently.

On Apr. 22, 1924, the child was sent for an X-ray study and the report was as follows: "Left mastoid does not show the presence of any bony destruction, necrosis or distinct bone involvement. A slight haziness is present, which would indicate an acute inflammatory condition, most probably exudative in character."

Urine, negative. White blood cell count, 15,000.

Operation: Following the usual routine, an incision 1 inch long was made in the crease behind the auricle, from below upward; free pus was welling up, pointing to the presence of a subperiosteal abscess. On clearing this away a fistulous opening was discovered in the mastoid bone posteriorly and below the antrum, leading into a mass of granulation tissue and extensively necrosed cells. The antrum was filled with pus and granulations blocking firmly the aditus ad antrum.

All this was thoroughly removed and the wound irrigated with a 1-8000 bichlorid solution and then packed with iodoform gauze. No sutures were applied.

On admission to the hospital and prior to the operation the child's temperature was 104,° pulse 110, and respiration 48; soon after the operation the temperature dropped to 100.1.° About 9 o'clock the same evening the temperature rose to 103.2,° the pulse jumped to 126, and respirations to 50. The rise in temperature was accounted for by a developing cellulitis, extending anteriorly as far as the left eye, closing it completely; downwards to the angle of the jaw, and posteriorly midway between the mastoid process and occipital protuberance. We removed the packing and applied boric acid dressings which were kept continuously wet. The temperature gradually dropped as the cellulitis cleared up and on the second day after the

operation the temperature was down to normal and the edema of the left side of the face and head had practically disappeared. The culture showed a streptococcus non-hemolyticus. The patient made an uneventful recovery, the wound healing in less than three weeks.

SUMMARY.

1. I believe that in every case of acute mastoiditis without apparent middle ear symptoms the infection must have at some time or other extended from the nasopharynx, by way of the Eustachian tube, and through the tympanum to the mastoid. The accompanying engorgement of the mucosa of the antrum and aditus ad antrum is at times sufficient to block the latter, and limit the infection to the mastoid, while the tympanic cavity symptoms may be so mild as to escape notice or be apparently absent.

2. Most cases reported as primary or atypical mastoiditis were atypical in the sense that the symptomatology was rather vague and the diagnosis was arrived at mostly with difficulty; but it seems that the greatest number of these cases were rather of the type of the "acute latent suppurative otitis media." The causative factor in a few of these must have been the "streptococcus mucosus," which was not recognized.

BIBLIOGRAPHY.

1. DABNEY, VIRGINIUS: Idiopathic Mastoid Abscess, *J. A. M. A.*, 65, 501-504; Aug. 7, 1915.
2. SALINGER, SAMUEL: Primary Periostitis of the Mastoid. Ann. Otol., Rhinol., and Laryngol., p. 943, Dec., 1920.
3. LEVI: Cong. Inter. d'otol. et Laryn., p. 33, 1889.
4. ERSNER, M. S.: Atypical Mastoiditis. *Atlantic Med. Jour.*, Oct., 1922.
5. HEMPSTEAD, BERT E.: Mastoiditis Without Apparent Involvement of the Middle Ear. *J. A. M. A.*, 81, 1266-1268, Oct. 13, 1923.
6. KING, CHARLES R.: Acute Mastoiditis Without Aural Discharge. *J. A. M. A.*, 81, 1523, Nov. 3, 1923.
7. AMSDEN, H. H.: Unusual Case of Mastoiditis. THE LARYNGOSCOPE, 34:134, Feb., 1924.
8. HEGGIE, NORMAN M., and KNAUER, W. J.: Mastoiditis Without Apparent Involvement of the Middle Ear. *J. A. M. A.*, 82: 1044, Mar. 29, 1924.
9. PUGNET, AMEDEE: Recurring Mastoiditis. Rev. de laryngol., Bordeaux, 45:166, Mar. 15, 1924.
10. BEEGER, HANS: Acute Atypical Mastoiditis (Without Perforative Acute Otitis Media). Acta Oto-laryngol., Stockholm, 7:87-97, No. 1, 1924.
11. WAGERS, ARTHUR J.: Acute Mastoiditis Apparently Primary in an Infant of Seven Months. THE LARYNGOSCOPE, 35:453-456, June, 1924.
12. DIEHL, J. E.: Mastoiditis-Epidural Abscess Without Apparent Middle Ear Involvement. Virginia Med. Monthly, 51:223, July, 1924.
13. BROWN, J. M.: Atypical Mastoiditis. THE LARYNGOSCOPE, 36:344, Aug., 1924.
14. GLEASON, E. E.: Atypical Mastoiditis. Manual of Diseases of the Ear, Nose and Throat, p. 496.
15. KOPETSKY and ALMOUR: Primary Acute Mastoiditis. THE LARYNGOSCOPE, p. 774, No. 10, Vol. XXXV.

1530 Locust Street.

SHOULD THE USE OF THE TERMS FIRST, SECOND AND THIRD DEGREE NYSTAGMUS BE CONTINUED?*

DR. GEORGE W. MACKENZIE, Philadelphia.

First, second and third degree have become the generally accepted terms for defining the intensities of vestibular nystagmus. The writer will attempt to prove the inaccuracy of expressing the intensity of nystagmus after this manner. However, let us first see what there is to commend it.

Everyone who has busied himself to any extent studying vestibular nystagmus must have observed the fact that the intensity of the nystagmus is increased when the subject manifesting it looks toward the side of the quick component; while the intensity is decreased when he looks away from it (toward the opposite side). This should not be interpreted as an indication that the vestibular reflex is actually increased when one looks toward his nystagmus and decreased when he looks away from it. The difference in the intensities is due to the introduction of an extraneous factor. Moreover, the character of this extraneous factor is well known. It is the physiologic nystagmus which all normal individuals manifest or tend to manifest when they look to the extreme side positions; in rare instances, also when looking upward. Some individuals manifest a more intensive physiologic nystagmus than others. With a few it is not manifested until they are commanded to increase the effort at side fixation. In other words, the intensity of this physiologic nystagmus is proportionate to the effort put into the attempt at side fixation. The plane of the nystagmic movements in the case of physiologic nystagmus is more or less in the plane of the action of the eye muscles called into play. For instance, when the average normal individual looks fairly intently to the right side in the horizontal plane, he will manifest a horizontal nystagmus to the right. With increased effort he will manifest a mixed form of nystagmus to the right; i. e., horizontal combined with rotary movements to the right. The rotary element (the quick movements *contra-clockwise* from the examiner's viewpoint) is due either to secondary action of the horizontally acting recti, or to the action of the obliques; or to the combined action of recti and obliques. In any case, the rotary element is present. If he looks still more intently to the right, he

*Read before the Philadelphia Laryngological Society, Jan. 5, 1926.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Jan. 18, 1926.

will manifest a pure type of rotary nystagmus to the right, in which there is not the least evidence of horizontally directed movements. Incidentally, the writer has never observed *clockwise* quick movements physiologically with the individual looking to the right.

While the plane of the nystagmus is changing, the intensity is also. To repeat then, italicizing the essential features. The average normal individual will, with *moderate effort* at looking to the right side, manifest a pure *horizontal* nystagmus to the right side, of *mild intensity*. With a *stronger effort* at looking to the right, he will manifest a *mixed horizontal rotary* nystagmus to the right, of *greater intensity* than the preceding horizontal form of nystagmus. With the *strongest effort* at looking to the right, he will manifest a pure *rotary* nystagmus to the right, *still more intensive* than the preceding mixed form of nystagmus.

All of these facts are important and must be borne in mind when studying cases with spontaneous nystagmus, whether it be of extra ocular eye muscle or of vestibular origin.

Since the plane of the eye movements and the intensity of the nystagmus are both regulated by the degree of angle deviation of the eyes from the straight ahead position, it follows that we have two variables to consider in determining the character and intensity of spontaneous nystagmus, the moment the patient's eyes are allowed to deviate from the straight ahead position.

To show the influence of the physiologic element, let us consider a case of recent right-sided labyrinthine suppuration, in which there is a pronounced nystagmus (mixed, horizontal and rotary) directed toward the left side. In such a recent destruction the spontaneous nystagmus directed toward the left side is so pronounced that it is manifestly evident even when the patient looks toward the right side, because the intensity of the vestibular factor, which is directing the nystagmus toward the left side, is stronger than the physiologic one which is tending to pull the nystagmus toward the right side. When the vestibular pull overbalances the physiologic, as in the above case, the intensity of the vestibular nystagmus is expressed in the term of the third degree.

Though the physiologic element in the case of third degree nystagmus is insufficient to overcome the vestibular, it, at least, has the power to inhibit the intensity of the vestibular manifestation—for, when the patient looks toward the right, the nystagmus to the left becomes less intensive than when he looks straight ahead or toward the left.

Take the same case of right-sided labyrinthine suppuration: After a week or ten days the vestibular nystagmus will have decreased ap

preciably in intensity, so that the physiologic pull to the right, when the patient looks to the right, balances the vestibular one to the left, when he will no longer manifest a third degree nystagmus. If spontaneous nystagmus to the left is observable when he looks straight ahead, then he is manifesting second degree nystagmus.

In not every case of second degree nystagmus does the physiologic pull to the right balance exactly the vestibular pull to the left, while the patient is looking toward the right. In some cases the physiologic pull to the right exceeds, somewhat, the vestibular to the left, with the result that the patient manifests a definite, though very slight grade of nystagmus to the right, if he looks intently enough in that direction.

Continuing with the same case of right-sided labyrinthine suppuration: A time comes, after several months, when the vestibular nystagmus to the left decreases so much that it is no longer observable, except when the patient looks to the left, that direction toward which the physiologic nystagmus is allowed the opportunity to pull in the *same* direction as the vestibular. If the nystagmus is manifested only so long as the patient looks toward the left side, it is referred to as one of the first degree, which is still weaker in intensity than nystagmus of the second degree.

To review it briefly: Spontaneous nystagmus of vestibular origin may manifest itself, according to its comparative intensity, as one of the first, the second or the third degree. The first degree is of the least intensity and its presence is indicated *only* when the patient looks toward the direction of his nystagmus. The second degree is more pronounced than the first degree and is manifested not only while the patient looks toward the direction of his nystagmus, but also when he looks straight ahead. A third degree nystagmus is the most pronounced and is not only manifested when the patient looks toward the direction of his nystagmus and straight ahead, but even when he looks away from the direction of his nystagmus. Furthermore, in a case of third degree nystagmus, let us say to the left side, because of a right-sided labyrinth destruction, or, in fact, any other affection capable of producing a nystagmus to the left side, the nystagmus to the left is most pronounced when the patient looks to the left, less pronounced when he looks straight ahead, and the least pronounced when he looks to the right.

All of this looks so easy and so simple, on the surface, that it naturally appeals to us, but let us look a little deeper. To begin with, a vestibular nystagmus, whether it is experimentally or pathologically produced, is a reflex manifestation, pure and simple, only so long

as there are no extraneous influences present. The least extraneous influence acts as a disturbing factor. The most disturbing factor to the free play of the vestibular reflex is the *voluntary* innervation of those eye muscles which are called into action by the reflex. Next, since the voluntary innervation of the extrinsic eye muscles is not constantly the same, we have a variable to deal with, comparable with that met with in the action of the ciliary muscle of the child, which is so bothersome to the refractionist. Again, the intensity of voluntary innervation differs considerably in different individuals. Besides, one in good health will innervate the eye muscles, in looking to the side position, two or three times as strongly as another who is materially below par in health, especially if the latter finds that looking to one side happens to increase the vertigo. Furthermore, if a patient happens to have a weakness of one of the extra ocular muscles, for instance, the right external rectus, he will manifest a more pronounced nystagmus to the right, when looking to the right, than he will to the left, when looking to the left, in the presence of a perfectly normal vestibular apparatus and pathways on both sides, in which case a tyro, using the popular method of examining and recording the findings, could very easily be misled into believing the patient has an irritative inner ear affection on the right side or a relative destruction on the left.

No one has a right to accept as evidential findings, in the case of a suspected labyrinthine involvement, the presence of rhythmic nystagmus, with the patient looking to one or the other side, and omitting the observations with the patient looking straight ahead. Besides, he must be able to determine the presence or absence of eye muscle weakness, or have it determined for him by someone else, capable of making the tests.

Let us next consider how these variables can modify the findings in the case of labyrinthine disease. With third degree nystagmus to the left, in the case of a recent right-sided labyrinthine suppuration, if the patient happens to be prostrated, as they usually are, with coexisting vertigo, he is in no mood to exert himself to any great extent. His vestibular element, therefore, is likely to suffer less neutralization from the play of his physiologic element than it is a day or two later, when the vertigo has become less intensive.

If for any reason whatever an individual develops a reflex vestibular nystagmus, for instance, to the left side, it can be observed perfectly well if one looks carefully for it, with the patient looking straight ahead at infinity. When he looks to the left side the nystagmus is naturally increased, because with the eyes directed toward the left, there are two influences at work, the vestibular reflex, plus

the physiologic nystagmus, a combination referred to earlier. The intensity of the vestibular reflex is a constant factor, but not so with the physiologic nystagmus. The latter is inconstant or variable Its intensity is in proportion to the effort put into the side glancing For instance, two observers examining the same patient, but a few minutes apart, may note a difference in the intensity of nystagmus, due entirely to the difference in the physiologic element, which has no connection whatsoever with the vestibular reflex; or it may even happen in the case of first degree nystagmus that one observer recognizes it, while another fails to. Thus two different opinions could arise concerning the real issue in the case.

First degree nystagmus, as a vestibular reflex manifestation, *cannot* exist independently. In some cases of supposedly first degree nystagmus, careful observation will reveal the presence of a nystagmus, with the patient looking straight ahead, in which case it must be placed in the class of second degree nystagmus. In some other cases of accepted first degree nystagmus, supposedly of vestibular origin, it will be found upon more careful study of the case, that the nystagmus is actually due to an eye muscle paresis. In the case of an imbalance of vestibular tonus from the two sides, from any cause, there will be manifested a nystagmus, slight though it be, directed toward the side of the greater tonus when the patient looks straight ahead.

The writer has pointed out on other occasions the differential diagnosis of nystagmus of vestibular origin and of external eye muscle paresis. In the case of nystagmus of vestibular origin the nystagmus must be evident when the patient looks straight ahead. In the case of nystagmus due to external eye muscle paresis, the nystagmus is evident only when the patient looks in that direction, which calls for the exercise of the weak muscle. It follows, therefore, that first degree nystagmus, pure and simple, can apply only to eye muscle conditions and is not applicable to labyrinthine conditions, and its continued use can lead but to confusion and inaccuracies.

We must take into account not only the influence of the physiologic nystagmic factor in modifying the intensity of the vestibular manifestation, at one time favoring and at another time inhibiting its full play, but we must consider also that other inconstant or variable factor of physiologic origin, which can also change the plane of the eye movements. For instance, a patient, because of a circumscribed irritative lesion in the right, external, semicircular canal, will manifest, all other things being equal, a pure form of horizontal nystagmus to the right, when he looks straight ahead. But if he looks intently to the right, the first thing which strikes one is the

increased intensity of the nystagmus. Next, it will be observed that the nystagmus is no longer purely horizontal, but it exhibits also a rotary element. Since mixed rotary and horizontal nystagmus, of pure vestibular origin, speak for a diffuse lesion and not a circumscribed one, it requires no stretch of the imagination to see how one can easily err in the diagnosis. The opportunity for mistakes is increased still more in the less intense processes. For illustration, we can take the reverse condition, that of circumscribed destructive lesion of the right, external, semicircular canal. To make it more difficult to diagnose, let us say that it is a latent process. In consequence, the patient manifests a slight degree of horizontal nystagmus to the left, with the patient looking straight ahead. Now, when he looks very decidedly to the left, he will exhibit either a mixed horizontal rotary nystagmus to the left, or even a pure rotary nystagmus to the left, depending upon the relative strength of the two factors, the physiologic and the vestibular. To add still further to the difficulties, it occasionally happens that supposedly normal individuals exhibit slight weakness of one or the other of the extra ocular muscles, which are elicited only with the red cover glass test, taken during extreme rotation of the eyeballs. If the weak eye muscle happens to be one of the obliques, which is normally brought into play with extreme rotation to the left, then it follows that in the case we are citing (latent, right-sided, circumscribed, destructive lesion of the external semicircular canal), the patient will manifest, on attempting to rotate the eyes to the left, instead of the expected pure horizontal form of nystagmus to the left, a rotary element out of proprtion to that found in the case of an individual with a normal eye muscle balance.

A more accurate method of recording the intensity of vestibular nystagmus is that which the writer has long since inaugurated and about which he spoke in discussing Dr. Metzenbaum's paper at the last meeting of the American Academy of Ophthalmology and Oto-Laryngology at Chicago, October, 1925.

If we desire to know the speed of a horse, what do we do? We consider two factors, time and distance. In other words, we figure out how much distance the horse can cover in a given time, or what length of time is required for the horse to cover a given distance. Following this same principle in the case of nystagmus, the greater the distance the eye moves, in a given time, the more intensive is the nystagmus. We can figure out the intensity of nystagmus by dividing the distance factor by the time factor. If we allow D to represent the distance; L, the length of excursions in millimeters; N, the number of excursion, then D=L times N. If we allow T to repre-

sent the time factor expressed in seconds, and I the index or intensity of the nystagmus, then $I=\frac{L \text{ times } N.}{T}$ For instance, if a patient with spontaneous rotary nystagmus of 1 m.m. length at the equator, occurring twice every second, then the intensity would be found as follows: Substituting $\frac{1}{2}$ m.m. for L, the numeral 2 for N, and the numeral I for T, then the intensity $I=\frac{1 \text{ times } 2,}{1}$ or 2.

In another case, where a patient manifests a nystagmus of 2 m.m. length, once every second, the intensity is found as follows: $\frac{2 \text{ times } 1}{1}=2$. The intensity in both these cases are the same, because the distance factor is the same in spite of the difference in the length of the excursions and the number of excursions.

Let us take the case of a very mild grade of nystagmus, found not uncommonly in the latent stage of labyrinthine suppuration, or during the subsiding stage of unilateral labyrinthitis of focal infection origin. The nystagmic movements occur but once every six seconds and then is only 1 m.m. in length; following the same method, substituting numerals for the algebraical letters, $\frac{1 \text{ times } 1}{6}=\frac{1.}{6}$ In this case the nystagmus is but one-twelfth as intensive as in the preceding cases.

One second of time is too short a period to observe the eye movements. In the last cited case, it required at least six seconds to make the observation. In a case of this kind it is well to make observations over a period sufficiently long as to permit one to see at least four excursions. For ordinary cases, however ten to fifteen seconds is a long enough period for observations. In any case, the time factor, measured in seconds, ticked off by the stop watch, is put down as the denominator. This method commends itself: *a*, because it is a method of observation that eliminates uncertain, extraneous factors, and *b*, it records the intensity of nystagmus after a mathematical method. It can be absolutely accurate only if the observations are made with a moving picture apparatus, a stop watch and suitable measuring instruments. However, one can learn by practice, without the aid of all these implements, to estimate the intensity of nystagmus within 15 per cent of accuracy, which is far ahead of anything thus far attempted.

1. First, second and third degree have become popular terms with the otologists for expressing the comparative intensities of rhythmic nystagmus, such as that found accompanying pathologic

RESUME.

conditions of the so-called vestibular apparatus, the vestibular nerve or its pathways.

2. It has this to commend it: That it is an attempt to express in simple terms the comparative intensities of nystagmus.

3. Nystagmus of first degree is the least intensive and is observable only when the patient looks toward his nystagmus, so to speak. Nystagmus of the second degree is more intensive than that of the first degree, and is observable not only when the patient looks toward his nystagmus, but also when he looks straight ahead. Nystagmus of the third degree is the most intensive of the three degrees and is not only observable when the patient looks toward his nystagmus and straight ahead, but also when he looks away from his nystagmus.

4. This method of recording the comparative intensities of nystagmus is not sufficiently accurate to serve the needs of science, since it introduces extraneous, inconstant factors or variables.

5. There is no such thing as rhythmic nystagmus of the first degree of vestibular origin, pure and simple.

6. What is referred to as first degree nystagmus of vestibular origin is either an overlooked second degree nystagmus of vestibular origin, or else a nystagmus due to an extraocular eye muscle paresis.

7. Inaccurate observations can lead only to false conclusions, which is sufficient reason for abandoning this popular method of recording the comparative intensities of nystagmus.

8. Vestibular nystagmus is a reflex phenomenon and can manifest itself in its purest form only when it is not hampered with the voluntary exercise of the same eye muscles which are brought into play by the reflex, just as one taking the patella reflex desires a complete relaxation of the extensors and flexors of the leg. Who would think of ascertaining the patella reflex with the quadruceps contracted or the hamstrings taut?

9. The best method of recording the intensities of nystagmus is one that comprehends the two essential factors, distance and time. Distance equals length of excursion times the number of excursions. This factor is then divided by time reckoned in seconds. The result can be accepted as an index of the speed or intensity of the nystagmus.

PERFORATION OF THE NASAL SEPTUM.

Dr. Louis Blumenfeld, Brooklyn, N. Y.

Perforation of the nasal septum has always been a worry for the rhinologist when operating for deviation or other abnormal condition of the nasal septum. While with care this complication can be avoided in most instances, still it does occur in spite of all precautions one may take.

For the repair of the perforated septum we must resort to some form of plastic surgery. Any operative procedure that will conserve tissue, diminish the area of granulating surface, and shorten the time of healing, is in my estimation of sufficient importance to be brought to the attention of the profession interested in this specialty.

The operative procedure I wish to bring to your attention is both simple and effective in its results. A minimum amount of tissue is transplanted, resulting in a minimum area of granulating surface. All the cutting is limited to one side of the septum. The granulating area is only on one side of the septum. The perforation is closed over by two layers of mucous membrane. No granulating surface that may result in sloughing is left at the site of the perforation.

In this particular operation, as in all plastic procedures, the preliminary treatment is just as important as the operation itself. The best type of surgery in an unprepared field will as a rule prove to be a failure. This is especially true in plastic surgery where the tissues must be in a healthful state and free from infection in order to insure a successful outcome.

The following is the routine treatment I have adopted as a preliminary to the actual operative procedure itself.

When a patient presents himself for the relief of symptoms produced by a perforated septum, he is carefully examined for any infective process present in his nasal cavities. If any are found they are treated until eradicated or until sufficiently under control so as not to be a menace to the operation or to the postoperative healing. Any ulceration found at or near the rim of the perforation is treated until epithelialized. The nasal chambers are kept free from crusts by appropriate treatment for a period of two weeks before operation.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Dec. 11, 1925.

As a result of the above preoperative care, the patient presents himself for operation with clean, healthy nasal chambers, and with every chance in favor of a successful outcome.

In preparation of the patient for operation the following routine is carried out: *1*, place 10 per cent cocain packs in the nasal chambers and wait 20 minutes; *2*, paint the outside of the nose and the adjacent facial region with half strength Tr. Iodin; *3*, remove the cocain packs; *4*, paint the nasal chambers with 2 per cent mercurchrome; *5*, inject ½ per cent novocain into the mucocutaneous junctions.

We are now ready to proceed with the operation. The steps follow: *1*, Make curved incision "a" as in Fig. 1. The length, curvature, and position of the incision depending upon the size, shape and position of the perforation. *2*, Elevate the mucous membrane of

Fig. 1 Fig. 2 Fig. 3

area "acd" in Fig. 1. *3*, Make incisions "b," "c," and "d." We now have a long flap "x" and two triangular flaps "y" and "z." *4*, Turn down the two triangular flaps through the perforation to the other side of the septum. *5*, By a little manipulation and easing up of the triangular flaps they can be approximated so that the apex of one triangular flap will be adjacent to the base of the other. *6*, In this position the triangular flaps should be sutured, the apex of one to the base of the other. The suturing is done on the other side of the septum, that is, the side opposite to where the incisions are made. *7*, Flap "x" is now lifted over the perforation and sutured to the elevated mucous membrane at "c" and "d." *8*, The only granulating surface left in the operation field is the area from which flap "x" was transplanted.

If there is any cartilage beneath flap "x" it is advisable to transplant some cartilage of approximately the same size and outline as the perforation with the flap when it is moved over to the site of the perforation.

The surgical procedure as outlined above is performed with extreme care and precision so as to make clean-cut incisions in order not to mutilate any of the flaps. After the incisions are made and the mucous membranes elevated they should be carefully deposited in their new positions without any undue tension and approximated with a minimum amount of suturing.

After this operation is completed, the septum is covered on both sides with sterile vaseline and the nasal chambers are lightly packed with sterile vaselined gauze. This packing is allowed to remain in the nose from 12 to 24 hours and then carefully removed so as to avoid disturbing the position of the transplanted mucous membranes.

The daily aftercare is to keep the nasal chambers clean, prevent crusts from forming on the septum by keeping it well vaselined. I emphasize the vaseline because it is important to prevent the irritating and drying effect of the air currents produced by breathing. The postoperative period usually lasts from ten days to two weeks. The sutures, which should be of black silk, are removed on the fifth day.

This procedure as outlined above has given me excellent results and I trust that any of my colleagues that may attempt this operation will find it equally efficient.

1564 St. Marks Avenue.

REPORT OF TWO CASES OF BRAIN ABSCESS PRESENT IN PATIENTS SUFFERING FROM NASAL ACCESSORY SINUS DISEASE.*

MAJOR ROBERT E. PARRISH, Washington, D. C.

Case 1: W. W., age 26 years, white. Family history negative. History of present complaint (from patient and transfer records): Always well until Apr. 5, 1924, when he became ill with a "heavy cold," headache severe, could not sleep, went to a hospital, became paralyzed on left side and then delirious, transferred to another hospital and had an operation on skull; an abscess was found in the right parietal region; this was drained. Paralysis, left side, cleared up. Headache improved. Second operation performed one month later. Under date of Apr. 5, 1924, a diagnosis of pansinusitis, acute suppurative was made. Under date of Apr. 11, 1924, diagnosis, abscess subdural, localized over fissure of Rolanda, secondary to ethmoidal sinusitis. At time of admission to this hospital and first seen by the writer, three and one-half months after onset of condition, patient had improved. Ears, normal. There was a herniation of the intercranial contents through the opening in the right side of skull. An examination of the nose and sinuses was made. Transillumination and X-ray showed definite evidence of involvement of the maxillary and frontal sinuses on the right side. There appeared to be most involvement in the right maxillary sinus. Patients' condition became rapidly worse, paralysis of the left side, came on a second time and the herniation of intercranial contents increased; patient's condition was hopeless. Died in a few days.

Autopsy revealed an extremely large abscess of the right cerebral hemisphere, which caused a herniation of this structure through the osteoplastically decompressed area.

The abscess cavity was filled with extremely thick, greenish-yellow pus. No other portion of the brain appeared to be involved except as a result of the increase in pressure. Autopsy limited to head. Autopsy by Major R. E. Scott, M. C.

Bacteriological examination: Pus from abscess, blood agar cultures, twenty-four hours; a long chain nonhemolytic streptococcus.

*These two cases are reported for the purpose of stressing the importance of nasal accessory sinus disease as a not infrequent cause of intercranial complications. This time, it is manifested by abscess formation, inside of the brain structure, and, in addition, subdural abscess in one case.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Nov. 28, 1925.

Post-mortem diagnosis: Abscess of brain, chronic, pyogenic, involving a greater portion of right cerebral hemisphere.

The sinusitis present when this patient came under observation was entirely on the right side—the same side as the brain abscess.

Case 2: J. W. K., age 34 years, colored. Admitted to hospital, July 8, 1924. Occupation, waiter. Family history negative. Personal history: Always well except for "trouble with nose and eyes." Never had any injuries. Had gonorrhea fifteen years ago. Eight years ago took a course of salvarsan.

History of present complaints: For the past five or six years has been "having trouble" with his eyes. Both eyes would "ache and pain." The vision in the right eye began to get bad about six years ago and has been getting worse ever since. Has an external deviation of the right eye, but does not know how long it has lasted. Is practically blind in the right eye. Has had a discharge of thick foul pus from the right nostril for more than a year. Never had any treatment for the eye or nose conditions.

Physical Examination: A well nourished adult, weight, 156. Eye examination: O.D., L. and S., A.S., 20/20. Divergent stribsmus nonparalytic, simple white atrophy, right eye. Nose and sinus, profuse purulent discharge present coming from right nares, foul odor.

Transillumination: Positive for involvement of right antrum.

Throat, tonsillitis chronic follicular bilateral moderate. Ears, normal. Hearing, normal.

X-ray Examination of Sinuses: "The frontal sinuses are large; no definite involvement. There is an increase in density over the right antrum." Puncture and lavage, right antrum, revealed the presence of a large amount of foul mucopus and caseous material. Case transferred from Eye Service to ENT service. Radical maxillary sinusotomy (Caldwell-Luc) performed Aug. 22. The sinus was filled with pyogenic membrane, pus and caseous material, with some polypi. Following this operation patient began to improve, the pain about the right eye began to grow less and the discharge from the nose practically ceased.

The general examination of this patient was negative except for some glandular involvement, principally on the left side of the neck. The blood Wassermann was double plus. Two doses of salvarsan was given before the maxillary sinus operation. Antileutic treatment later continued. Patient continued to improve. The glandular enlargement began to subside after the second antileutic treatment. On Sept. 16, while waiting to receive the weekly antileutic treatment, patient had an attack which was considered an epileptic seizure. The attack lasted about one-half hour and then patient ap-

parently recovered. One week later patient was feeling so well that he asked to be discharged from hospital, but was told that his treatment was not sufficiently advanced to justify this. An ethmoid operation, right side, was contemplated. Patient refused to have spinal puncture. Sept. 25, patient woke up complaining of a headache and had a temperature of 100.° The first time he had had any fever since admission, except following each dose of antileutic treatment. About 1 p. m., had another attack, which appeared at first to be an epileptic seizure. Passed into a state of coma and died less than one hour later. Five minutes before death, respiration, 12; pulse, 140.

Autopsy report by Major R. E. Scott, M. C. Head: "Extremely thick scalp exposing normal calvarium. After removing an especially thick calvarium even for a negro, a normal appearing dura is revealed. There is no apparent internal pressure. On removing the dura no evidence of hemorrhage. Vessels are not especially enlarged. The frontal lobe of the right hemisphere of the cerebellum is moderately adherent in its fossa. While removing the brain an abscess cavity is ruptured, which involves the temporal lobe, right cerebrum. On opening the ventricular system, both lateral ventricles of normal size and contain extremely turbid fluid. The third ventricle is apparently normal, as well as the fourth. There is no gross evidence of hemorrhage. There is a distinct localized abscess approximately 2 c.m. in diameter, in the temporal lobe of right cerebrum and the brain structure for a distance of 4 or 5 c.m. surounding this is extremely soft and necrotic, with an extremely offensive odor. Examination of the base of the skull reveals a dark area of the dura over the ethmoidal plates. On opening this, ethmoidal cells are filled with necrotic tissue throughout, with sequestra of bone and emitting a very foul odor, connects with the nares on the right side. The sphenoid sinuses are apparently not involved. This is an apparent infectious process, does not appear to be malignant and is not grossly suggestive of syphilis."

Diagnosis: 1. Brain abscess old, infectious, involving temporal lobe, right cerebral hemisphere.

2. Ethmoiditis chronic right. (Maxillary sinusitis previously operated and considered cured.)

Bacteriological examination: pus from abscess showed pure culture, B. Pyocyaneus. In the X-ray examination the brain abscess could not be seen and the diseased area extending up from the posterior ethmoid region could not be visualized. The sinusitis, maxillary and ethmoid, was present on the same side as the brain abscess.

Walter Reed General Hospital.

LIPOMA OF THE PHARYNX.*

DR. F. R. HERRIMAN, New York.

Lipoma of the pharynx is very rare. In the 1907 edition of Kyle's "Diseases of the Nose and Throat," the statement is made that but one case had been reported. This, however, is incorrect, since as far back at 1849 Foucher (Union Medicale) had reported a lipoma implanted on the anterior wall of the pharynx near the upper orifice of the larynx. Altogether, about fifteen cases had been reported by 1907. A pretty thorough examination of the literature has revealed reports of twenty-five cases of lipoma of the *pharynx*, exclusive of fifteen reports of lipoma of the *larynx* collected severally by Shattock (Proc. Roy. Soc. Med., 1909), and by Goldstein (THE LARYNGOSCOPE, 1909).

The present series of twenty-five cases of pharyngeal lipoma is about equally distributed between males and females, and covers a range in age extending from 4 to 92.

Symptoms of pharyngeal lipoma vary somewhat according to the location of the growth: According to Magne (Rev. hebd. Laryngol., Otol., et Rhinol., 1908) benign tumors of the inferior posterior wall betray their presence by esophageal signs, as dysphagia, regurgitation of food, repeated vomiting, sometimes accompanied by momentaneous appearance of the tumor in the buccal cavity and even outside. Tumors of the anterior wall have the same signs as those of the base of the tongue, i. e., laryngeal phenomena, troubles of respiration, phonation, accidental introduction of food in the larynx, etc. Tumors of the lateral walls have mixed symptomatology, both esophageal and laryngeal. Magne further states that generally speaking, pedunculated tumors are more annoying that sessile; the more voluminous, than the smaller. Nevertheless, very small tumors can provoke grave troubles, and the physical and moral condition of the patient may be affected; extreme emaciation and inanition may result.

"These tumors are of insidious beginning, and functional troubles appear little by little with increase of the neoplasm. Usually for tumors located on the posterior and the lateral walls, a volume equal at least to that of a nut is necessary before notable difficulty occurs in pharyngeal functions."

*Read before the Section of Laryngology and Rhinology, New York Academy of Medicine, Oct. 28, 1925.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Dec. 12, 1925.

In the series of cases under consideration, the point of implantation included various locations in the pharyngeal walls, as well as the soft palate, tonsil, and supratonsillar fossa. Sonnenkalb (Zeitschr. f. Ohrenheilk, 1916) believes that the lateral pharyngeal walls are the site of these fatty tumors more frequently than the posterior walls. It is difficult to state definitely of some growths whether they are pharyngeal or laryngeal as structures of both locations may be involved. As regard lipoma of the tonsil, Onidi (Rev. hebd. Laryngol., Otol., et Rhinol., 1899) states that up to 1895 there had been no reference to such a growth, and no one knew whether such a phenomenon was possible. Four cases of lipoma of the tonsil are included in this group. Avellis (Arch. f. Laryngol., 1898) thinks that fat cells must have been isolated in the tonsil in embryonal life.

St. Clair Thomson (1917) states that dipoma of the pharynx (which he notes as extremely rare) is nearly always connected with the soft palate, where it occurs as a smooth, regular or lobulated ovoid growth, yellowish in color and conveying the suggestion of fluctuation or of a tense cyst. It may also occur under the mucous membrane of the posterior wall of the pharynx, or it may grow from the supratonsillar fossa. Some of these growths are long and pedunculated, hanging loose in the pharynx and larynx, occasionally being coughed up by the patient.

Positive diagnosis is generally very easy. Analysis of the characteristics of the growth, its consistency, palpation of ganglions, examination of general conditions, usually give an impression of the precise nature. In doubt, biopsy.

Etiology: In attempting to account for the presence of lipoma in the fossa of Rosenmuller, Lawner (Monatsch. f. Ohrenheilk., 1913) argues:

1. That fat growths may be formed either from fat tissue or from other forms of connective tissue.

2. That misplaced fat buds stand in causal relationship with the development of these lipomas.

Shattock (1909) in connection with his work on lipoma of the larynx, took normal larynges from cadavers of obese individuals and examined sections of different laryngeal sites to determine the possible presence of fat cells as a normal phenomenon. In all locations examined he found fat cells in connection with the mucous glands and in the interglandular connective tissue, and about the cartilage. He states that fat cells may be normally met with in all positions from which these different laryngeal lipomata have been found growing all extrinsic. While no one has examined the pharynx for

the presence of fat cells, it seems reasonable to suppose that they would occur here normally much as in the larynx. The occurrence of submucous fatty tissue in general has long been recognized. Both Rokitansky and Virchow refer to submucous lipoma of the stomach and intestines (Shattuck).

As regards *cellular pathology,* pharyngeal lipomata show the characteristics of lipomata elsewhere in the body. In this series of twenty-five cases, there were nine in which the microscopic examination showed pure lipoma in all specimens examined; others were noted as pure lipoma in certain portions of the growth and fibroma in other portions; still other tumors were reported as lipomyxofibroma. Feaser (Elinb. M. J., 1916) thought that in his case the growth was a fibroma becoming myxomatous while the myxoma was turning into a lipoma. There have been a very few cases in which the pharyngeal lipoma was supposed to be recurrent after incomplete enucleation of dipoma of neighboring structures, as the parotid glands. Incomplete removal would quite probably lead to recurrence, and possibly to eventual evolution of malignant characteristics, although there has been so far as known, no such case reported.

Prognosis in pharyngeal nonmalignant tumors is generally good, although intervention is sometimes laborious and subject to surprises. Treatment is surgical. Pediculated tumors are removed by cold snare or galvanic loop; sessile tumors are removed by dissection. Pharyngolomy is reserved for very rare cases which are inoperable by the buccal route. Without operation, the patient is in danger of death from strangulation. In Holt's case (Trans. Path. Soc., Lond., 1854) the patient, a man, age 80 years, died suddenly while smoking his pipe, presumably from sudden cough and displacement of the growth, which was found, post-mortem, to be a pendulous fatty tumor, 9 inches long, filling the pharynx and extending downward toward the esophagus. Taylor (Trans. Path. Soc., Lond., 1877) reports the death of a girl, age 4 years, after two ineffectual punctures of a supposed pharyngeal abscess, followed by emergency tracheotomy. Post-mortem examination showed a lipoma in front of the upper cervical spine, but behind the pharynx.

In most of the cases here considered, simple excision or removal by cautery snare has sufficed to extirpate the tumor, although a number of operators have been forced to do a preliminary tracheotomy. Cheval (Bull. Soc. Roy. Sci. Med., Brux., 1902) was forced to do this by the size of the growth, as was also Laurens (Arch. Internat. Laryngol., Otol., et Rhinol., 1904), whose patient de-

manded a general anesthesia. This case was followed by a secondary necrosis of the hyoid bone with suppuration, necessitating hyoidectomy. Kellner (Monatschr. f. Ohrenheilk., 1909) removed a fibrolipoma, size of a walnut, springing from the fossa of Rosenmuller, very easily through the nose with the cold snare. A unique case was reported by Hannecart (Ann. Soc. Belge de Chir., 1900) in which a multiple lipoma of the anterolateral pharyngeal wall reached from the level of the inferior maxilla to the clavicle, the volume of a child's head. These multiple growths were removed through an incision along the anterior border of the sternocleidomastoid muscle; they were encapsulated and adherent to one another by their envelopes. Tilley (Proc. Roy. Soc. Med., 1919) removed a large submucous lipoma of the palate and pharynx under "open ether," but difficulty of oral respiration necessitated laryngotomy. The operation was identical with that for tonsil enucleation by dissection.

In the case to be reported, the patient, a man, age 68 years, complained of difficulty of deglutition, inability to eat solid food. He believed that his palate was too long. He had also suffered from deafness for four months. On examination, a swelling was found, the size of an English walnut, at the level of the soft palate, extending from the midline of the posterior pharyngeal wall ¾ of an inch onto the right lateral wall, and 1¾ inches downward. Two Roentgen ray examinations were negative for abnormality of the cervical spine. Wassermann report was also negative. Clinical diagnosis, lipoma of the pharynx. Operation, Apr. 23, 1925. Under local anesthesia, an incision was made down the median line of the growth, and an encapsulated tumor was dissected out. Hemorrhage was controlled by adrenalin and pressure; there was practically no hemorrhage during operation. Wound closed by suture without drainage. Healing by pimary intention.

Microscopic examination showed the growth to be a lipoma, with fat cells of large adult types. The pathologist noted in his report that lipomata in this region are rare.

BIBLIOGRAPHY.

APLAVIN, M. A.: Zur Technik der Pharyngotomia subhyoidea. Ein Fall von Lipoma durum im Cavum pharyngo-laryngeale enfernt per pharyngotomiam subhycideam. Arch. klin. Chir., 1891, xli, 324-336.

BRUNS, V. N.: Neue Beobachtungen von Polypen des Kehlkopfes, Monograph, Tuebingen, 1868, p. 154.

FARLOW, J. W.: A Case of Polypoid Lipoma of the Larynx. *N. Y. Med. Jour.*, Nov. 16, 1895, lxii, 610.

GOLDSTEIN, M. A.: Lipoma of the Larynx. THE LARYNGOSCOPE, Sept., 1909, xix, 641-670.

HINKEL, F. W.: Report of a Case of Fibro-lipoma of the Larynx. Trans. Twentieth Annual Meeting, American Laryngological Assn., May 16, 1898, p. 75.

HOLBECK, M. K.: Lipoma multiplex arborescens aditus ad laryngem. Protik. Zasiedamia. Imp. Kavkazsk. Med. Obshtsh., 1891-92, xxviii, 422-427. Also in the "Atlas" of Seifert and Kahn, Wiesbaden, 1895.

HOLT: Fatty Pendulous Tumor of the Pharynx and Larynx. Trans. Path. Soc., London, 1853-54, v., 123-125.

JONES, S.: A Fat tumor of the Larynx. Trans. Path. Soc., London, 1881, xxxii, 243.

KOEHLER: Ueber Lipom des Kehlkopfes. Reported by Seifert, Sitzungsb phys. med. Gesellsch. zu Wuerzbourg, 1894, 32035. Also in the "Atlas" of Seifert and Kahn, Wiesbaden, 1895.

MCBRIDE, P.: Clinical Notes on Fatty Tumors of the Larynx. *Edinb. M. J.*, Feb., 1889, xxxiv, 703-707 (two cases).

SCHROETTER, V. L.: Lipoma des Kehlkopfes. Vorlesurogen ueber die Krankheiten des Kehlkopfes, 1893, p. 271.

SHATTOCK, S. G.: A Large Laryngeal Lipoma of the Epiglottis and Base of the Tongue, with a Collection of Submucous Lipomata of the Intestine and of the Larynx. Proc. Roy. Soc. Med., Path. Sect., 1909, ii, 285-296.

TOBALD: "Laryngoscopie," Berlin, third edition, p. 436. Cited by v. Schroetter in Krankheiten der Kehlkopfes, 1892, i, 270.

WAGNER, E.: Die Todesfalle in der letzen Pockenepidemie von Leipzig. Arch. f. Heilk., 1872, p. 108.

OTT: Paris thesis, 1880, No. 28 (reports Richet's case of lipoma of soft palate).

RIPAULT: Un Cas de Tumeur Volumineuse du palais. Ann. Mal. Oreille, Larynx, etc., May, 1898, xxiv.

ROE, J. O.: Fibro-lipoma of the Pharynx. *J. A. M. A.*, Sept. 29, 1894, xxxiii, 491-492.

SONNENKALB: Ein pendelndes Fibrolipom des Rachens. Zeitschr. f. Ohrenheilk., 1916, lxix, 261-263.

TAYLOR, F.: Fatty Tumor Behind the Pharynx. Trans. Pathol. Soc., London, 1877, xxviii, 216-218.

TILLEY, H.: Large Submucous Lipoma of Palate and Pharynx. Proc. Roy. Soc. Med. (Sect. Laryngol.), 1919, xii, 189-190.

ZOLKI, L.: Ueber ein congenitales Fibrolipom der Gaumentonsille. Zeitsch. f. Ohrenheilk., 1903, xliv, 3.

OTHER ARTICLES ON PHARYNGEAL TUMORS.

BEESTEN, V.: Rachengeschwulst. Zentralbl. f. Chir., 1909, xxxvi, 1484.

FURSTENBERG, A. C.: Report of Two Cases of Thyroadenoma of the Posterior Pharynx and Nose. *J. Mich. St. M. Soc.*, Apr., 1918, xvii, 146-148.

LAWNER, S.: Geschwulste in der Gegend des Ostium pharyngeum tubae. Monatssch. f. Ohrenheilk., 1913, xlvii, 262-272.

MAGNE, P.: Un cas nouveau de tumeur benigne du pharynx inferieur. Rev. hebd. Laryngol., Otol., et Rhinol., Apr. 4, 1908, xxviii, 401-406.

PIETRI: Tumeur benigne du pharynx. *J. Med. Bordeaux*, 1908, xxxviii, 234-235.

WISHART, D. J. G.: Tumor of the Hypopharynx in a Child of Eleven Weeks. *N. Y. M. J.*, Mar. 16, 1921, cxiii, 458-459.

LITERATURE OF LIPOMA OF THE PHARYNX.

AVELLIS, G.: Lipom der Mandel. Arch. f. Laryngol., 1898, viii, 560-561.

BACH, J. A.: A Report of Two Cases of Large Sublingual Dermoid Cysts; One Case of Fatty Tumor of Pharynx and Nasopharynx. Med. Age, 1891, ix, 298-299.

BOND: Large Lipoma of Soft Palate. Proc. Laryngol. Soc., London, Nov. 4, 1898, vi, 8.

CHEVAL: Lipoma pharyngien volumineux. Extraction par les voies naturelles. Guerison. Bull. Soc. Roy. Sci. Med. Bruxelles, 1902, lx, 77-79.

FINDER, G.: Ein weiterer Beitrag zu den Lipomen der Gaumenmandel. Arch. f. Laryngol. und Rhinol., 1904, xv, 159-160.

FOUCHER, E.: Polype lipomateux insere sur la paroi posterieure du pharynx; nouveau prodede operatoire. Union med., 1849, iii, 290.

FRASER, J. S.: Fatty Tumor of the Hypopharynx Obstructing the Larynx. *Eding. M. J.*, 1916, xvi, 45-49.

GAUDIER, H.: Lipome du voile du palais. Bull. Laryngol., Otol., et Rhinol., 1909, xii, 1-5.

HANNECART: Lipomes multiples parapharyngiens et polypes du pharynx. Ann. Soc. belge Chir., 1900, viii, 315-317.

HAUG: Lipo-myxo-fibrom der Mandel. Arch. f. Laryngol., 1896, iv, 269-271.

INGALLS, E. F.: Fibro-lipomatous Tumor of the Epiglottis and Pharynx. *N. Y. M. J.*, Dec. 9, 1899, lxx, 840.

KELLNER, E.: Beitrage zur Histologie der gutartigen Guschwulste des Rachens und Nasenrachenraumes. Monatsschr. f. Ohrenheilk., 1909, xliii, 91-97.

KENYON, E. L.: A Large Lipoma of the Laryngopharynx; Removal Extraorally Under Cocain. *J. A. M. A.*, June 17, 1911, 1793-1794.

LABAT: Paris Thesis, 1874, No. 355 (reports Ollier's case of lipoma of the soft palate).

LAURENS, G.: Pharyngotomie Transhyoidienne Pour Un Volumineux Lipome de L'Epiglotte. Arch. Internat. Laryngol., Otol., et Rhinol., 1904, xviii, 38-40.

MILLIGAN: Proc. Laryngol. Soc., London, Jan. 1902, ix, 41 (pharyngeal lipoma) cited by Shattock, Proc. Roy. Soc. Med. (Pathol. Sect.), 1909, ii, 285-296.

ONODI, A.: Lipome de l'Amygdale. Rev. hebd. Laryngol., Otol., et Rhinol., June 17, 1899, xix, 696-699.

TONSILLAR INFECTION AS A CAUSE OF MENIERE'S DISEASE.*

DR. JUDSON DALAND, Philadelphia.

During the past five years, more and more attention has been directed towards the relationship of focal infection to the auditory apparatus. The reports of cases by George E. Shambaugh, George W. Mackenzie,[1] William G. Shemeley,[2] Francis E. Emerson[3] and others, clearly show that vertigo, tinnitus, and deafness may be secondary to focal infection. In children, the infection is usually located in the tonsils or adenoids, and in adults, in the tonsils or teeth.

The patient, a male, age 64 years, who was first examined in April, 1924, could only remain under observation for a short time prior to departing for Mexico, where he now resides; it was therefore impossible to make frequent clinical observations. As the otologist lost his notes, detailed description of the various equilibrium and other auditory tests could not be obtained.

The chief symptom was attacks of severe objective vertigo, the first occurring in 1922, and lasted three or four days; the second, about a week later, and continued ten days. The third attack in February, 1923, was accompanied by nausea, and lasted about ten days. In each of these attacks, although hungry, the thought, sight, taste or smell of cooked food was nauseating.

The last attack of vertigo resembled those observed in Meniere's disease, which he described as "terrific," objects moving swiftly in a vertical and rotary manner, necessitating rest in bed.

He furthermore described mild attacks of vertigo, which could always be induced by turning the head to the right, by bending backward, by suddenly assuming a horizontal position, or when suddenly placed in a recumbent from a sitting position while in a barber's chair.

He also complained of bilateral tinnitus aureum, more marked in the left ear, weakness, emaciation. He was pale, irritable, irascible, and only feebly resistant to cold.

He had uncomplicated scarlet fever at the age of 2 years; rickets at the age of 6 years; dengue fever at the age of 31 years; infected cervical glands were removed at the age of 32 years; nervous ex-

*Read before the Philadelphia Laryngological Society, Jan. 5, 1926.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Jan. 13, 1926.

haustion at the age of 47 years, and stiffness of the neck, relieved by the extraction of the teeth, at the age of 58 years.

He was markedly psychoneurasthenic, and has suffered from a series of shocks, due to the death of his mother, an unfortunate marriage and business reverses. The patient is easily fatigued, mentally depressed, worries from trifling causes, uses tobacco moderately, alcohol occasionally, leads a sedentary life, and has never had tonsillitis.

Physical examination reveals emaciation, adynamia, small tonsils, anterior pillars congested, arteriosclerosis, myocardial weakness, vertigo induced by a sudden change in position, tinnitus aureum, impaired hearing, the loss of many teeth, and markedly exaggerated knee jerks.

The erythrocytes were 4,690,000, or 93 per cent; hemoglobin, 90 per cent; leucocytes, 7,000; polymorphonuclears, 60.5 per cent; large lymphocytes, 10.5 per cent; small lymphocytes, 27.5 per cent; eosinophiles, 1.5 per cent. Blood pressure observations and urinalyses normal.

As the anterior half arches were deeply congested, and the blood showed a moderate grade of lymphocytosis, and as no other focus of infection could be discovered, infected tonsils were suspected. Dr. Ross H. Skillern reported on Apr. 18, 1924, that both tonsils were atrophic and contained pus, and cultures from the tonsils after removal, showed many streptococcus viridans, with a few staphylococcus albus.

He was under the care of physicians in Magdalena and Santa Fe, New Mexico, and skilled internists in Cleveland and New York City.

On Mar. 18, 1924, a laryngologist diagnosed Meniere's disease, due to otosclerosis, more pronounced in the right ear, and reported chronic inflammation of the right ear, with perforated drum, which was greatly retracted and sclerosed. The ticking of a watch could not be heard on either side unless pressed against the ear. No nystagmus.

Another skilled physician was of the opinion that arteriosclerosis involving the cerebrospinal vessels, in connection with stress and strain was the cause of the vertigo.

The diagnosis was Meniere's disease, chronic tonsillar infection, cardiovascular sclerosis, psychoneurasthenia, otosclerosis, impaired hearing, and tinnitus aureum.

Immediately after the tonsils were removed by Dr. Skillern, on May 5, 1924, the vertigo induced by turning the head and change of position of the body, entirely disappeared.

Autogenous vaccine and sodium iodid was advised, and he departed for a ranch in Mexico. Six months later, lymphocytosis disappeared.

Occasionally, during the past seventeen months, transitory attacks of slight vertigo occurred, apparently due to nervous exhaustion, but during the past five months, there has been *no* vertigo.

Remarks: The assumption that Meniere's syndrome and vertigo on movement of the head and body was due to irritation of the vestibular branches of the eighth nerve, secondary to toxins formed by the streptococcus viridans in the infected tonsils, is based upon the following reasons:

1. That no attack of Meniere's syndrome has occurred since the tonsils were removed.

2. That vertigo on movement of the head or body, which has been present for two years, disappeared *immediately* after the tonsillectomy, and only occasional mild and transitory attacks occurred during the succeeding year, and none during the past five months.

3. That no treatment was employed excepting an autogenous vaccine and 5 gr. sodium iodid three times daily.

The absence of lymphocytosis in November, 1924, indicated the absence from the blood of toxins from the streptococcus viridans.

The infected cervical glands, removed at the age of 32 years, were probably due to infected teeth and tonsils.

REFERENCES.

1. "The Report of a Case of Neuritis of the Eighth Nerve, Involving Both Branches from a Focal Infection in the Appendix," by Dr. George W. Mackenzie, Annals of Otology, Rhinology and Laryngology, June, 1918.

2. "Reports of Additional Cases of Neurolabyrinthitis," by Dr. William G. Shemeley, *The Journal of Ophthalmology, Otology and Laryngology*, April, 1922.

3. "Conduction Apparatus in the Prognosis of Deafness," by Dr. Francis E. Emerson, Annals of Otology, Rhinology and Laryngology, Vol. 30, p. 527, 1921.

4. *Ibid.*, p. 995.

5. "Perception Deafness," *Ibid.*, p. 1006.

AGRANULOCYTIC ANGINA. REPORT OF A CASE WITH FRACTURE OF THE TIBIA.*

DR. ROBERT J. HUNTER,** Philadelphia.

In the last three years, about a score of cases have been reported, with the diagnosis of agranulocytic angina, agranulocytosis, leukemic condition with marked leucopenia, malignant leucopenia with low neutriphil count and similar titles. These cases have all had a peculiar blood picture, combined with lesions in the throat. As laryngologists we will be called to see these cases. It is for this reason that I present this subject tonight.

The onset is usually abrupt, with the symptoms of an acute infection such as high fever, chill, headache, malaise, nausea, pain in the throat, teeth and bones. The cases have been in females aged 22 to 65 years. As the disease progresses, the throat becomes red and ulcerations, either single or multiple, appear. The tonsils are usually involved and at times the cheeks, tongue, gums, or larynx. There is a dirty, white, fibrinous exudate, upon the tonsil and surrounding affected areas, resembling diphtheria. This has been seen involving the superficial layers of the mucous membrane in some cases, but in some the tonsil was necrosed, so that it looked as if it had been enucleated. In one case the tip of the tongue necrosed, as if about to separate, before death intervened. The odor of the breath is marked. Similar necrotic processes have been found in the vagina, intestinal tract and skin. A brawny induration of the skin of the left breast, and in our case an erythema multiforme, have been noted. The authors stress the fact that there is no tendency to hemorrhage into the skin, but in one case which corresponded with this picture in other respects, there were petechia. Icterus has been seen frequently.

The blood findings are the characteristic diagnostic feature. The red blood cells, hemoglobin and blood platelets are normal or slightly reduced. The white blood cells are markedly reduced to as low as 600. The polymorphonuclear leucocytes are greatly reduced or even absent, thus giving us the name, agranulocytosis. The lymphocytes and large lymphocytes are relatively increased, to as much as

*Read before the Section on Otology and Laryngology, College of Physicians of Philadelphia, Oct. 21, 1925.

**From the Department of Surgery, Division of Laryngology, Philadelphia General Hospital.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Dec. 7, 1925.

100 per cent (actually diminished, on account of the low white count). Transitionals are about 4 per cent. In some of these cases due to the difficulty in finding them, less than a hundred cells were counted in making the differential. The bone marrow shows a distinct decrease in white cells, with few or no granulocytes or myelocytes and many lymphocytes. The erythroblasts and erythrocytes are in normal number.

The Wassermann is negative; blood culture usually negative, but the streptococcus hemolyticus and the bacillus pyocyaneous, have been found. The latter was found in the throat and spleen after death in two cases. The coagulation time and the bleeding time are normal. Cultures from the throat have been negative for diphtheria and have shown the usual flora. Smears have been negative for Vincent's organisms, with but one exception. The urine has shown hyaline and granular casts, but no leucin or tyrosin.

Further general examination of the patient reveals no characteristic findings. Leon reports three cases with some enlargement of the spleen and liver and three without. The glandular system is not involved. The temperature range is 103° F. to 105° F. The course is from three to fourteen days and is usually fatal. One case has recovered and one has had a fatal recurrence in two years. Death has usually resulted from pneumonia.[1 2]

Report of a Case: On May 10, 1925, K. F., a married woman, age 45 years, was admitted to the surgical ward of the Philadelphia General Hospital. The patient stated that on May 8 she tripped, fell and hit her left knee.

Past Medical History: She had milk leg eighteen years before, followed by varicose veins. Two years before, these broke down. She came to this hospital with blood poisoning of the leg. The leg was incised. She stated that she had had epilepsy since childhood.

Sex History: Married at 19 years. Five pregnancies; no still births or miscarriages. Menopause seven years before with no trouble. Removal of one ovary and suspension of uterus, twenty-three years before. Married twice. She has lived with present husband twenty-four years.

Physical Examination: The patient was a middle-aged, rather obese, adult female, lying in bed with complaint of pain in the left leg. Head, negative. Mouth and throat, strong odor of alcohol, otherwise negative. The tongue protruded in the middle line, with some slight tremor at the tip. It was heavily coated. Both jaws were toothless. The nose and ears showed no apparent discharge or obstruction. The chest was of a large, obese, emphysematous type, with pendulous breasts. The percussion note was somewhat im-

paired at the right base, where scattered rales were heard. Otherwise the lungs were normal. The apex of the heart was in the fifth interspace, midclavicular line. There were no murmurs or thrills. The aortic and pulmonic second sounds were accentuated. The abdomen was markedly pendulous. No pain or tenderness was elicited. The liver and spleen were neither palpated nor percussed, beneath the costal margins. The inguinal glands were not palpable. The genital organs were not examined. The pulse was equal on both sides, easily collapsible; frequency, 100. The respiration was 26; the temperature, 99° F. The upper extremities were negative. The biceps reflex was normal. The right leg showed a normal knee jerk, with no ankle clonus or Babinski. The left leg was markedly swollen from the toes to the hip. The left knee was very large, discolored and fluctuating, as if filled with fluid. Scars of previous incisions were seen.

Provisional Diagnosis: *1.* Fracture of the left tibia. *2.* Cellulitis of the left leg. *3.* Epilepsy. *4.* Acute and chronic alcoholism

Notes: May 11 and 20, urine negative. May 11, white blood count, 5,900 cells; hemoglobin, 9.6 gm.[4] Polymorphonuclear leucocytes, 66 per cent; lymphocytes, 30 per cent; transitionals, 4 per cent. Wassermann, negative to 2 antigens. From May 10 to May 20, the temperature averaged 99° F., with the highest 101° F., immediately before operation. X-ray showed a badly comminuted fracture of the base of the tuberosity of the left tibia. The shaft of the bone had been impacted; another line of fracture went upward into the joint. The proximal end of the fibula was fractured. May 12, an epileptic attack. May 17, Buck's extension and Thomas splint applied. May 20, fracture set by Dr. Hearn, with nitrous oxide and ether anesthesia. May 28, nurse's note: Sleeps well, no complaint. At this point the temperature went to 102° F. for the first time and the patient complained of headache and pains all over the body. It became apparent that she was suddenly much worse. On May 30, a fine rash was seen on the chest and arms. It was thought to be a dermatitis, due to the intensely hot weather we were having at that time. This progressed in the next 48 hours, to a typical case of erythema multiforme. There were no hemorrhages into the skin or mucous membranes, nor any areas suggesting necrosis of the skin. On June 1, the patient complained of sore throat, which rapidly grew worse. On June 5, the ears and nose were normal. The soft palate, uvula, and the anterior pillars of both tonsils were eroded and red. The superficial layers of the mucous membrane were involved and the line of demarcation between the healthy and diseased mucous membrane, was abrupt without any

surrounding area of erythema. There was no exudate. The tongue and the posterior wall of the pharynx were dry. There was some diffuse redness of the hard palate. Examination by Dr. Boston the same day, revealed fine crackling rales, at the end of inspiration, with some impairment on percussion, at the base of the right lung, suggesting bronchopneumonia. On June 5, the blood count was: red cells, 3,200,000; hemoglobin, 8.1 gm. per c.c.; polymorphonuclear leucocytes, 0; lymphocytes, 89 per cent; large lymphocytes and transitionals, 5 per cent; unclassified, 2 per cent. A later count the same day showed white blood cells, 1,000; hemoglobin, 8.1 gm. per c.c.; polymorphonuclear leucocytes, 0; lymphocytes, 93 per cent; large lymphocytes and transitionals, 5 per cent; unclassified, 2 per cent. A final count by Dr. Pons was: white blood cells, 800; polymorphonuclears, 0; lymphocytes, 95 per cent; mononuclears, 5 per cent. On June 6, the blood culture was negative; there were no tubercular bacilli in the sputum, no Vincent's organisms in the smear from the throat; there were pneumococci, staphylococci, and influenza bacilli in the throat culture; a heavy trace of albumin, a few hyaline and no granular casts in the urine. After May 29, the temperature range was from 101° F. to 105° F. The patient died on the evening of June 6th. Permission for autopsy was refused.

COMMENT.

This disease is apparently a distinct entity, although it has also been classed with the leukemias. It may be due to an acute infection. In sections of tissue, vast numbers of bacteria have been found. This may be interpreted, however, as a result of the great reduction of polymorphonuclear leucocytes, rather than as a causative factor. Injection of guinea pigs with bacillus pyocyaneous has indicated that this organism will cause a leucopenia, with a marked reduction in the percentage of neutrophils.

In our case, the fracture and cellulitis may have been causative factors, by opening the tissues to infection. On the other hand, they may have been a coincidence. Seyderhelm[3] quotes Burger's case of acute hemolytic icterus, following a fracture of the forearm. In this instance, either the erythrocytes are destroyed more quickly than the marrow can supply them, or the erythropoetic function of the marrow is depressed. In agranulocytosis a similar process is seen, except that the granulocytes are involved. In purpura hemorrhagica, the platelets are diminished, and in aplastic anemia we have all the elements of the blood, greatly reduced.

Cases of obscure ulceration of the throat, septic sore throat and obstinate Vincent's angina should always have a complete blood count taken.

SUMMARY.

Agranulocytic angina is characterized by ulceration of the throat and other organs, with a very low white blood count, showing few or no polymorphonuclear leucocytes.

The reported case was preceded by injury to the long bones.

BIBLIOGRAPHY.

1. See LEON, ALICE: Deutsch. Arch. f. klin. Med., 143:118, Aug., 1923, for bibliography of collateral reading.
2. See MOORE, J. A., and WIEDER, HENRY S.: *Jour. Am. Med. Assn.*, 85: 512, Aug. 15, 1925, for bibliography to date.
3. SEYDERHELM: Deutch. Med. Wchnschr., 50:1362, Oct. 3, 1924.
4. Multiply by 7 to get percentage.

2011 Chestnut Street.

ACUTE LARYNGEAL EDEMA.*

DR. DAVID N. HUSIK, Philadelphia.

Acute laryngeal edema is not a distinct disease, but rather a complication, secondary either to a local or systemic infection. By acute laryngeal edema, we mean an inflammation, and infiltration of the mucosa of the larynx, and its cartilages, with embarrassed respiration.

Edema of the larynx, especially involving the epiglottis, and aryepiglottic folds is a commoner condition than most of us are lead to believe. If we were to make a routine examination of the larynx, in all infectious diseases, especially influenza, scarlet fever and typhoid, and in all inflammatory diseases, about the upper respiratory tract, such as peritonsillar abscess, acute tonsillitis, Vincent's angina, and deep abscesses around the neck, we would be surprised to find that edema of the larynx is not an unusual condition. Mild cases with hoarseness, and slight pain in the larynx are often overlooked, and only accidently discovered during a routine examination of the upper respiratory tract.

There is no relationship between the severity of the primary infection, and laryngeal edema. A mild primary infection may cause severe laryngeal edema, as was demonstrated in the late influenza epidemic. Patients with mild forms of influenza had severe laryngeal inflammation with marked edema of the larynx, greatly embarrassed respiration, and in a few reported cases, death.

*Read before the Section of Otology and Laryngology of the College of Physicians of Philadelphia, Jan. 20, 1926.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Feb. 1, 1926.

Clinically, edema of the larynx may be divided into two classes: *1* Acute inflammatory or infectious type; *2* non-inflammatory or non-infectious type.

Acute inflammatory or infectious type may be subdivided into two groups: *a* Those secondary to abscess formation or infection of faucial tonsils, as in peritonsillar abscess, acute tonsillitis, secondary to deep infection in the neck, and abscess of lingual tonsil; *b* those occurring during infectious diseases, such as scarlet fever, influenza and typhoid.

Noninflammatory or noninfectious type include edemas caused by foreign bodies, burns, passive congestion from heart and kidney conditions, certain drugs, such as potassium iodid, edemas from constitutional diseases, such as T. B. and lues, and last under this class might also be mentioned the angioneurotic edemas.

Case Reports. Case 1: A young lady, age 25 years, was admitted to the University Hospital for a tonsillectomy. The operation was performed under ether anesthesia, very little bleeding, and patient returned to bed in good condition. Next morning she complained of a very severe pain on the right side of the throat, and did not feel like getting up, so the interne decided to keep her in bed another day. Towards evening I was called in on account of severe pain and some difficulty in breathing. Upon laryngeal examination I was very much surprised to see some edema of the epiglottis and aryepiglottic folds, right side, with a good deal of edema of the posterior pillar on the same side. I ordered ice by mouth, ice-collar, cold adrenalin spray directly applied to the larynx, and to notify me as soon as dyspnea gets worse. Midnight I received a telephone call that the patient is having respiratory difficulties, and is bluish. I was over at the hospital in a few minutes, and the larynx at this time showed complete infiltration of the epiglottis, patient sitting up and gasping for breath. I performed a tracheotomy, which gave her immediate relief. The next day the patient felt better and was breathing quietly through the tube, and the laryngeal edema was much quieter. The swelling of the larynx gradually disappeared, and on the fifth day, I removed the canula. The patient made a good recovery, but rather an unusual experience following tonsillectomy. The only history we got from the patient was that she was subject to acute attacks of tonsillitis, having gotten over one of these attacks a few days before operation.

Case 2: E. R., colored, age 30 years, was admitted to my service in the Philadelphia General Hospital, June 9, 1924, with a diagnosis of edema of the glottis, and the following history: June 6, or three days before admission, she had a suspicious looking throat, and

was given 25,000 units of antitoxin. Two days later she developed a peritonsillar abscess, which was opened by Dr. Mitchell, a former interne of the hospital, who referred the case. The throat was negative for diphtheria and Vincent's. On the morning of June 9 she began to have respiratory difficulties, and was sent to the hospital that afternoon. I saw the woman about 4 p. m. She was in a sitting posture, her head was thrown back, she had an anxious expression, and was breathing with great difficulty. Examination of throat showed a typical appearance of a peritonsillar abscess, on right side, and a large opening through the soft palate, where an attempt was made to open the abscess. Probing around there for a few minutes failed to discover any pus. She had a great deal of swelling of the right side of her neck, and could barely open her mouth. Laryngeal examination was performed with difficulty, the epiglottis was inflamed and highly infiltrated, occluding a deeper view of the larynx. Tracheotomy was immediately performed under novocain anesthesia. The patient returned to bed in good condition. Notes on the charts are as follows:

7 p. m.: Patient breathing very much better, and is sleeping quietly.

10 p. m.: Resting very well, breathing is quiet and regular.

12 m.: Resting easy, temperature 101,° tube changed and cleansed.

June 10, 1:30 p. m.: Patient was seen by my assistant, Dr. Schwerin, and reported that she was doing very well, breathing quiet and was able to swallow, and seemed in every way comfortable.

2:15 p. m.: The nurse noticed that the patient's breathing became irregular, and when the interne came over five minutes later she was dead.

Clinical Diagnosis: Edema of the glottis, pulmonary embolus. Gross anatomical lesion, sixth month pregnancy, dilatation of the heart, pulmonary edema, acute parenchyneatous nephritis, toxic fatty change in spleen, cystic adenoma of thyroid. Larynx ulceration and infiltration at the base. Mucosa alone involved. Vocal cords negative. Acute suppurative cellulitis, large pitutary. Liver congested.

Cause of Death: Acute suppurative sellulitis, following opening of peritonsillar abscess, of course eclampsia and septicemia might also have been a potent factor, as the cause of death.

Microscopic Description of the Brain: Sections from representative areas of the brain show as follows: the pia-arachnoid shows no abnormalities, the vessels within the pia are not thickened. The cortex shows definite edema with widening of the individual layers. No pervascular infiltration is present. In the cerebellum there is also edema. Microscopic diagnosis: edema of the brain.

Tuley in the *Kentucky Medical Journal*, reports a fatal case of laryngeal edema following influenza. The patient had suddenly gone to the bad, and he found the patient in a sitting posture in bed, his expression was anxious, he was bathed in cold perspiration, and his breathing was so noisy that it was difficult to distinguish anything definite on stethoscopic examination of the chest. Preparations for a tracheotomy were made, but before the laryngologist arrived fifteen minutes later the patient was dead.

Autopsy findings: The trachea was incised downward to the bifurcation. The lower portion showed considerable congestion of the mucosa, and there was marked edema of the glottis on the left side. The mucosa and submucosa from the epiglottis and aryepiglottic fold being swollen, puffy, exuding an amber colored fluid. This case was undoubtedly one of acute laryngeal edema, following influenza, and had an immediate tracheotomy been performed the patient's life might have been saved.

Edwin D. Smith, of Louisville, reports a case of influenza, in which examination shows slight cyanosis, flushed cheeks and anxious expression. There was no cough, the lungs were negative, no sign of Ludwig's angina, or infection in the oropharynx. Examination of the larynx showed swollen epiglottis. Since he had seen three cases, with same symptoms, die, he decided to do a tracheotomy. The patient immediately inhaled deeply through the tube, and his face became pink. On third day following operation, his general appearance was very good, and swelling practically subsided; on the fifth day the tube was removed and the patient made an uneventful recovery.

Acute laryngeal edema following the noninflammatory or noninfectious type as a rule clear up under local measures. On several occasions I have been obliged to perform tracheotomy on a T. B. and luetic edema of the larynx. The angioneurotic edema--by angioneurotic edema we understand an acute edema of the larynx in a neurotic individual, with no etiology except neurosis. I must admit that I have never seen an edema of the larynx of this type; that is, I have never seen laryngeal edema, that I could not see some local or constitutional cause for it.

Inherited nervous conditions are important etiological factors in these classes of edemas. Dr. Osler has reported a case in which the disease occurred in five generations, of thirty-nine members of the family, nineteen had angioneurotic edemas, two dying of edema of the larynx. He states that edemas may be brought about by some toxin in the intestinal tract, and in a certain percentage of cases the larynx is the first part to be attacked.

1610 Spruce Street.

THE BRONCHOSCOPIC TREATMENT OF LUNG SUPPURATION WITH SPECIAL REFERENCE TO THE POST-TONSILLECTOMY TYPE.*

DR. M. C. MYERSON,† New York.

The increased number of lung abscesses recognized in the past few years has awakened a real interest in this subject. The larger number of tonsillectomies performed and the recent influenza epidemic are probably the two greatest contributing causes to this condition. This paper deals with the general subject of the bronchoscopic treatment of lung suppurations. The post-tonsillectomy type of abscess will receive special consideration because it is of particular interest to the laryngologist.

Of interest and importance in the consideration of this type of abscess, is the method of causation and the question of whether this unfortunate sequel cannot be avoided entirely, or to a great extent, A knowledge of the symptomatology and course of the condition is of importance in order that the operator may know how to recognize this sequel when it occurs. And, lastly, a consideration of the treatment of these lesions, and the advantages of the various types of treatment is of value. It is now fairly well known that a few hundred post-tonsillectomy abscesses have occurred since Richardson[1] and Manges[2] first reported their cases. The history is usually the same; the individual is tonsillectomized, usually under ether anesthesia, and four to twenty-two days later he has a chill, pain in the chest, a dry cough and an elevation of temperature. At this stage an erroneous diagnosis of pneumonia might be made, but in the course of the next few days he begins to expectorate a foul smelling sputum. The profuse expectoration of foul odor usually makes the diagnosis. In very young children the pus is frequently swallowed and there is no expectoration. There is however present a distinct odor to the breath, as a rule. When the odor is not detectable, dependence must be placed upon the roentgenogram. The roentgenogram taken when the expectoration has begun shows a fluid level if the cavity is not entirely filled at the time the film is taken. A fluid level in a lung cavity is positive evidence of abscess.

Although opinions are not in agreement as to the method of causation of these lesions, it is fair to state that a majority of those

*Read before the Section on Laryngology, New York Academy of Medicine, May, 1925.

†Associate Laryngologist, Mt. Sinai Hospital.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication July 3, 1925.

interested in the subject favor aspiration as the route of infection. In a previous study it was shown that 155 of 200 patients tonsillectomized under general anesthesia aspirated a varying amount of blood, mucus, and crypt content during the operation. These patients were followed up one month after operation, and there was not a single case of proven pulmonary lesion among them. It was therefore reasoned that it was not the aspiration that influenced the production of a lung abscess, but rather the inability of a given portion of the lung to rid itself of aspirated material. It was put forth that post-influenzal, tuberculous, and some post-pneumonic changes predispose to fibrosis in the parenchyma and smaller tubules; that this fibrosis meant an absence of resiliency and elasticity, and that therefore the particular area involved was an area of non-expulsion.

A suspicious chest must be taken into consideration if a tonsillectomy is to be performed. Aspiration into a damaged pulmonary field must be avoided.

When the laryngologist experiences the embarrassment of an abscess complicating his operation, he becomes very much concerned about his responsibilty in the matter, and asks himself and others if there is any way in which he might have avoided it. It is fairly well conceded that patients who are operated upon under local anesthesia and are wide awake, do not as a rule, aspirate blood and mucus as a result of the operation. A very small percentage do, but this percentage is closer to 3 or 5 than the 77½ of the general anesthesia cases. Prevention then would lie in the choice of that anesthetic which gives the lesser incidence of aspiration. In adults, therefore, local anesthesia should be the method of choice. It might be added here that the use of cocain for swabbing the pharynx is not necessary and can be dispensed with, so that possible inhibition of the laryngeal reflex by anesthetization of the superior laryngeal nerve can be avoided. Novocain infiltration gives ideal anesthesia for these operations in the hands of many laryngologists. In children too young for a local anesthetic, who have known or suspected pulmonary lesion, the writer would advocate light anesthesia and some method of causing the patient to cough immediately after the operation. This coughing would insure the evacuation of the tracheobronchial tree and lung bed. Such a method would be the irritation of the larynx by an instrument such as the tongue depressor or suction tube. It has been demonstrated in a previous study that the material aspirated during tonsillectomy does not afford a sufficient stimulus to cause cough in the presence of a light anes-

thesia, and that those patients who coughed did so because of pre-existing catarrhal tracheobronchitis, or of a catarrhal tracheobronchitis caused by the forced administration and concentration of the ether, or because of the contact of an instrument with the laryngeal structures.

If a patient unfortunately develops an abscess it is the desire of all concerned to rid him of his trouble as quickly as is possible. The cure of a lung abscess depends upon collapse and obliteration of the cavity. To obtain this end there are two types of treatment, the operative or nonexpectant, and the nonoperative or expectant. Under the nonoperative or expectant plan of treatment we have a purely medical supervision of the patient. He is given fresh air, sunshine, good food, postural drainage, perhaps vaccines and exposure to the rays of the alpine lamp. Under the operative or nonexpectant plan of treatment we include any procedure that will hasten the recovery of the patient, either by the production of drainage and spontaneous collapse, as is done in bronchoscopy, or by the forced collapse, as is done by artificial pneumothorax or extrapleural thoracoplasty. Artificial pneumothorax is not suitable for peripheral abscess because of the danger of rupture into the pleural cavity; further, there is no assurance of cure with this method. However, one of the ten cures of post-tonsillectomy abscess here recorded was due to artificial pneumothorax. The surgical procedure for collapse is a fairly extensive one; here also the results have not shown a high percentage of cures. Therefore a milder substitute that will acomplish as much or more deserves consideration. The more radical treatment of these conditions, which has not yet been mentioned, consists of extirpation of the diseased area or lobe by the knife or cautery.

In acute abscesses bronchoscopy is of value because by thinning out the exudate with the irrigation, and by evacuation of this exudate with the suction apparatus, the cavity can be kept emptied for a sufficiently long time to permit of a spontaneous collapse and obliteration of the cavity. The bronchoscopic irrigation and aspiration of lung abscess has gained many advocates since Yankauer first introduced his method some ten years ago. His procedure was based upon the fact that the patient had an area of involvement in the lung from which it was difficult or impossible to expel the exudate; that usually the exudate was so tenacious and so viscid that it would not come away unless it were thinned out and removed by aspiration. There is one type of abscess in which it seems wise to omit irrigation and use only aspiration. I refer to the early acute

abscess which may or may not have become localized. If nature has not placed a leucocytic barrier around the process, irrigation should be omitted for fear of spreading the inflammation. Even when nature has placed her barrier in early cases, this barrier is so delicate and slender that it is better to simply aspirate so as not to disturb it. In acute abscesses the exudate is as a rule much thinner than it is in abscesses of longer duration, therefore the factors of viscosity and adhesion do not enter so much into consideration as in the older cases. It is in the subacute and chronic abscesses that irrigation is of advantage and necessary to thin out the pent up exudate so that it may be properly removed. One of the bronchoscopic cures in the writer's series was obtained after a single bronchoscopic aspiration of the left upper lobe bronchus.

While it is true that a fair percentage (40 per cent in this series) of post-tonsillectomy abscesses heal spontaneously, it is nevertheless true that bronchoscopic treatment would accomplish those same cures at a much earlier date, in other words, if bronchoscopy were permitted the duration of the illness would be appreciably shortened. The accompanying tables give an analysis of fifteen lung abscesses following tonsillectomy. The first table is self-explanatory. The second shows six, or 40 per cent, of spontaneous cures, and three, or 20 per cent, of bronchoscopic cures. One case was cured by artificial pneumothorax. All of these cures were obtained in acute cases, or in cases that were just passing the acute stage. Attention is invited to the fact that in only three of these cured cases was bronchoscopy permitted. The duration in the spontaneously cured cases could most likely have been markedly decreased if permission had been obtained for bronchoscopy. If it is true that a majority of post-tonsillectomy abscesses are caused by aspiration, what would be more rational and more logical than that a bronchoscope and an aspirating tube be introduced as near to the area of suppuration as is possible, and that the involved area be ridded of its pathologic content? The fact that a large percentage of these abscesses recover under the usual medical care is no reason why these individuals should be allowed to suffer many more weeks than is necessary. Bronchoscopy has a definite aim and accomplishment in these cases. It is my feeling that bronchoscopy in early abscess offers almost certain cure in the light of our present knowledge. There is no element of certainty when these cases are treated medically. Further, those cases treated by the expectant plan beyond a period of six or more weeks will be more difficult to cure than those of shorter duration. When drawing conclusions from the tables of the post-

tonsillectomy abscesses here presented, it must be remembered that in only three of the cases of short duration was bronchoscopy permitted. All of these patients were cured. In two of these three the duration was less than three weeks, and in each of these cases evidence of cure was obtained within less than two weeks from the time of the initial bronchoscopy. In the third abscess, which was of three months duration, cure was much more difficult because of the loss of time; here twenty-eight irrigations were required, for the patient no longer had an acute process. It was rather a subacute process tending toward chronicity. The advantage of early bronchoscopic treatment in these cases cannot be too strongly stressed. The earlier a case is seen, the better is the chance for cure.

If the bronchoscopist were given the opportunity to treat these acute cases, it is ventured that there would be fewer chronic abscesses. In cases of chronic abscess the bronchoscope can accomplish a great deal. Roughly, a chronic abscess might be considered one in which definite tissue changes have taken place. There is a dilatation of the smaller bronchial elements, granulation tissue, and some fibrosis, with more or less fixation of the cavity wall. Inasmuch as cure of a lung abscess depends upon the complete obliteration of the cavity, it is readily manifest that fibrotic changes in the periphery will interfere with a cure. It is conceivable that a very considerable number of irrigations frequently carried out in such an abscess, might by means of healthy granulations and replacement fibrosis, eventually produce a cure; but this would be the rare exception. Only a specific for the type of infection in the individual case could cure the type of abscess just considered. Such a specific would have to keep the cavity sterile so that healthy granulations could fill it up. Such a specific might be found in bacterotherapeutic methods of which the bacteriophage solution is an example.* Even then, if we should have a fixed cavity which has become sterile, we must always face the possibility of future reinfection.

Of interest to the attending physician in these chronic cases is the fact that we can make a majority of these patients comfortable. The cough is lessened in frequency, the expectoration is reduced in amount, the odor disappears, the clubbing of the fingers recedes, and the patient is much more comfortable and active. When such a result is not obtainable with the bronchoscope, the patient should be turned over to the surgeon. Most surgeons prefer not to operate

*The writer in association with Dr. Bullowa and Dr. G. Schwartzman was able to obtain a recovery in a lung abscess of eight months' duration by the employment of a specific bacteriophage solution in his bronchoscopic irrigations.

upon patients who are doing well, and are comfortable under a method of treatment that is simpler and a lesser hazard than surgery.

The bronchoscopic irrigation is carried out with no anesthesia in children and a very little local anesthetic in adults. The bronchoscope is so placed that the combined irrigating and aspirating tube can be introduced into the smallest approachable bronchial branch from which pus can be seen coming. While the solution is being slowly introduced, the suction apparatus is withdrawing the wash fluid through the aspirating tube. The irrigation is not performed under pressure; the fluid is permitted to flow in very slowly and gently. Iodin solution, saline, and various nonirritating antiseptic solutions have been used. The mechanical cleansing of the area would seem to be the principal factor, rather than any antiseptic action of the introduced fluid.

In closing it should be said that neither bronchoscopy nor any other type of treatment for lung abscess is the method of choice in these cases. Co-operation between the internist, the surgeon, the roentgenologist and the bronchoscopist should be the decided influence in the treatment. It is important that the profession understand that bronchoscopists do not claim that they have a cure all.

TABLE ONE

Analysis of Fifteen Posttonsillectomy Lung Abscesses.

Ages	3 to 45							
Lobes	Right Upper	2			Left Upper			3
	Right Lower	9			Left Lower			1
	Appearance of first symptoms after operation.							
		4 to 22 days.						
	Spontaneous	6	R. L.	5	L. L.			1
Cures	Bronchoscopic	3	R. L.	2	L. U.			1
10	Pneumotharax	1	R. U.	1				
Improved	Bronchoscopic		(chronic)	1	R. L.			
	Medical		(acute)	2	R. U.	1	L. U.	1
Unimproved	Medical		(acute)	1	R. L.			
Death	Medical		(acute)	1	L. U.			

TABLE TWO

Cures of Posttonsillectomy Abscesses in Relation to Duration of Symptoms.

		Spontaneous.				
	Sex	age	duration			lobe
H. A.	M	45	10 weeks			R. L.
M. M.	F	32	6 weeks			R. L.
N. S.	M	19	5 weeks			R. L.
M. S.	M	11	7 weeks			R. L.
F. D.	F	30	3 weeks			L. L.
L. D.	F	7	5 weeks			R. L.
		Bronchoscopic			No. bronchoscopies	
M. H.	F 30	12 weeks		R. L.	28	
T. K.	M 4	13 days		R. L.	2	
N. L.	F 3	3 weeks		L. U.	1	
		Pneumothorax				
G. D.	F	12	6 weeks			R. U.

30 East 40th Street.

LOW PERFORATION OF TRACHEA FROM GUNSHOT WOUND, WITH REPORT OF CASE.

DR. THOMAS J. CARMODY, Danville, Ill.

Subcutaneous rupture or perforation of the trachea, with subsequent subcutaneous emphysema, is a rare accident. A careful search of the literature discloses that there have been fifty-four cases reported to date; therefore, I thought that an additional case would be of sufficient importance to be reported. Zeuch, in 1922,[1] tabulated fifty-three cases; this tabulation was made after a thorough search of the entire world's literature. Mortality of all cases was 54 per cent. The autopsy cases showed that death was due to air and blood in the mediastinum, causing suffocation, although contributing crushing injuries had a bearing upon the mortality. Nixon reported an additional case in October, 1925.[2] This case was a spontaneous rupture of the trachea and with the three previously reported in Zeuch's tabulation, made four reported cases of spontaneous rupture of the trachea. A tracheotomy was performed, with recovery.

The authorities agree that some sort of an outlet must be established for the escaping air, the choice between a simple incision down to the trachea wound, a tracheotomy, or an actual suturing of the injured trachea, is a subject for debate. Theoretically, an effort might be made to suture both the point of entrance and exit of the trachea, or if only one opening is present, closing the same might be accomplished, however, in any case drainage should be established for some time.

Case Report: Mar. 12, 1925, male, age 17 years, single, was admitted to the hospital, complaining of pain and difficulty in breathing since injury a few hours previously. Ten hours previously was shot in the neck; while standing on the landing of the stairs, at the foot of the stairs a young lady friend jokingly pulled a revolver from the pocket of a boy friend's overcoat which she was wearing, and pointing it at him, standing three steps above, fired point-blank towards him. At the time he felt a stinging pain in the neck and consulted a physician, who advised him to go immediately to the hospital for attention. This he declined to do until the following morning.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Dec. 15, 1925.

Examination, Mar. 12, 1925, 9 a. m.: Well nourished young man, face flushed. Bullet hole present just above the episternal notch, precisely in the median line. Deep pressure at the base of the neck elicitated the peculiar crepitus bubbling, showing the presence of free air in the subcutaneous tissues. The emphysema seemed to be confined to the base of the neck.

X-ray of the chest discloses: "a small circular shadow of increased density about the size of a .32 bullet, which is located opposite the inferior margin of the third dorsal vertebra on the right side.

Fig. 1. The lateral view shows bullet located in the superior mediastinum, just opposite the mid-portion of the third dorsal vertebra. X-ray examination by Dr. W. H. Miner.

In the lateral view this opaque shadow seems to be located in the superior mediastinum and is located just opposite the mid-portion of the third dorsal vertebra on the right side, which is shown very clearly in the lateral view."

Fluoroscopic examination of the esophagus: "The heart and aorta seem to be normal in size and shape and the diaphragm seems quite regular on both sides and the posterior mediastinum appears clear. On the administration of an opaque meal the esophagus ap-

pears to be normal in size, shape and position and I see no evidence of an abnormal opening through the wall of the esophagus at this point. On fluoroscopic examination there seems to be a small circular shadow of increased density about the size of a .32 bullet. This is located just opposite the inferior margin of the third dorsal vertebra on the right side, and is located quite far back posterior near the spine."

Examination, Mar. 12, 1925, 4 p. m.: The tissue at the base of the neck is now swollen as well as the soft tissue through the upper

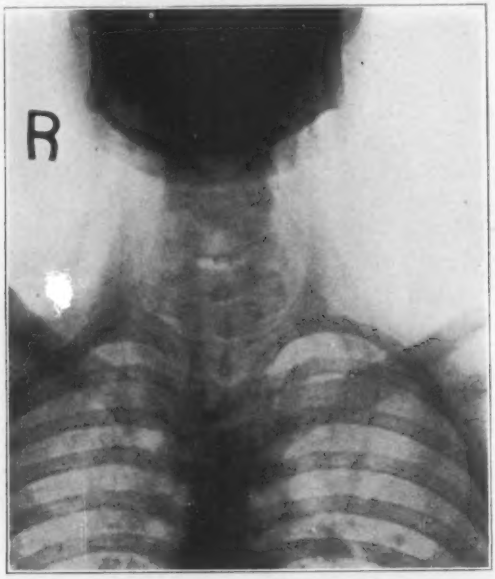

Fig. 2. View shows small circular shadow of increased density located in line with the inferior margin of the third dorsal vertebra on the right side. X-ray examination by Dr. W. H. Miner.

part of the chest and neck. Subcutaneous emphysema can be detected throughout the body. Temperature, 101.4°; pulse, 140; respirations, 42. The heart sounds were peculiar, having a metallic sound. Patient was now in great distress. The respirations were labored, due, of course, to the tremendous pressure against the bronchi and trachea from the large amount of air in the mediastinum and in the base of the neck.

Diagnosis: Perforation of the trachea.

Indications for operation: A patient with a leaking trachea and increasing pressure in the mediastinum, against the heart and neighboring tissues.

Operation performed at 8 p. m. A low incision was made down to the episternal notch, under nitrous oxid gas anesthetic. On account of the tremendous swelling it was difficult to locate the trachea quickly. The point of entrance was located about 2 inches below the level of the upper border of the sternum. There was a continual flow of air from this wound. Incision was made into the

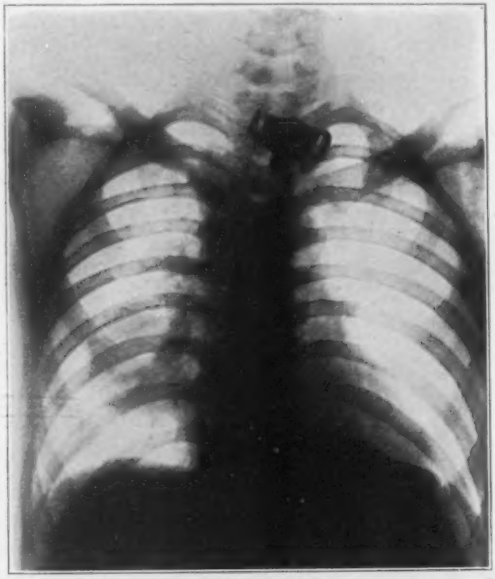

Fig. 3. X-ray showing lower lobe consolidation, also considerable widening of the mediastinal space. X-ray examination by Dr. W. H. Miner.

trachea and a tracheal canula inserted. On account of the depth of the wound it was necessary to enclose the canula within the wound itself; this was done with silk sutures.

Patient slept well on the first night. On the fifth post-operative day the emphysema had disappeared. On the sixth post-operative day the patient had a chill, with rise in temperature and pulse.

X-ray of chest at this time shows that the "heart seems somewhat increased in its transverse diameter and the mediastinal shad-

ow remains widened to about the same degree as shown by the last Roentgenogram taken previous to this date.

A homogeneous density involving the right middle and lower lobes which I believe is due to consolidation such as we get with a pneumonic process. The upper lobe of the right lung seems quite clear and in the second interspace, upper lobe bronchus shows a marked amount of peribronchial thickening involving the medium-size bronchioles, but this infiltration does not extend out to the periphery of the lung.

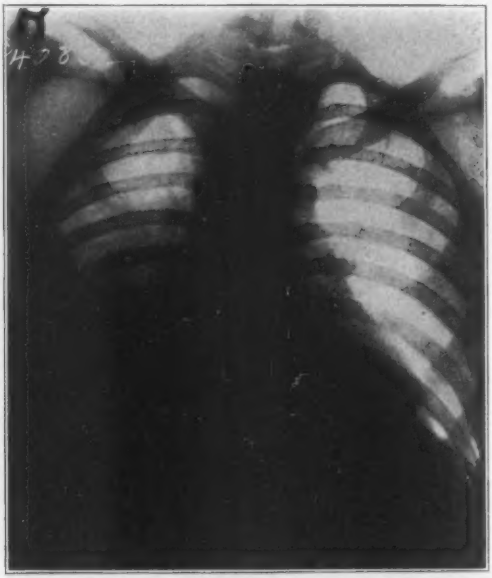

Fig. 4. Well developed consolidation of both the middle and lower lobe; heart increased in its transverse diameter. X-ray examination by Dr. W. H. Miner.

Summary of X-ray: A moderate amount of haziness of the middle and lower lobes of the right lung, which I believe is due to consolidation, and the interlobar pleura is slightly thickened, which is due to an interlobar pleuritis.

Urinalysis: Quantity of urine passed in 24 hours, 2300 c.c. Color, clear. Specific gravity, 1016. Reaction, acid. Albumin, negative. Sugar, negative. No casts or pus cells found.

Blood: Hemoglobin, 90 per cent. Red cells, 3,600,000. White cells, 14,000. Polys., 57 per cent. Small monos., 30 per cent. Large

monos., 6 per cent. Eosinophiles, 2 per cent. Transitional cells, 4 per cent.

Diagnosis: Lobar-pneumonia.

The duration of this atypical type of pneumonia was four days. The tracheal tube was removed on the twenty-eighth post-operative day. There has been no evidence of tracheal stricture. The patient has experienced no pain from the presence of the bullet and his appearance is that of a perfectly normal individual in every way.

BIBLIOGRAPHY.

1. Zeuch, L. H.: Subcutaneous Rupture of the Trachea. *Ill. Med. Jour.*, Vol. XLI, No. 6, June, 1922, p. 451.
2. Nixon, J. W.: Rupture of the Trachea." *J. A. M. A.*, Vol. 85, No. 16, Oct. 17, 1925, p. 1221.

408 The Temple.

AN UNUSUAL CASE OF ACUTE INFECTION OF THE EPIGLOTTIS AND LARYNX; TRACHEOTOMY AND RECOVERY.

Drs. Louis Daily and N. A. Allen, Houston, Tex.

J. G., age 8 years. Family history negative. He is one of two healthy children; father and mother living and well.

Previous history: Had measles and whooping cough; had his tonsils and adenoids removed four years ago.

Present history: Came home from school complaining of feeling badly and sore throat, and was brought to our office at 4 p. m. on Dec. 4. He had a general hyperemia of the mucous membrane of the throat; the epiglottis was not swollen; he had no fever; he was sent home with an astringent gargle and the diagnosis of a mild pharyngitis.

That evening he refused his supper, complaining of pain on swallowing, and Dr. Allen saw him at 8 o'clock in the evening; the child still manifested no alarming symptoms. Temperature 101.°

At 9 o'clock the next morning the child was seen in bed, breathing heavily, and having the typical pinched face of difficult respiration. On examination of the throat the epiglottis stood out as a round red swelling. His temperature was 102,° pulse 100, respiration 36.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication, Oct. 23, 1925.

He was sent to the hospital for a puncture of the epiglottis and ice applied to his neck in the meantime.

When he was brought into the operating room at 11 a. m., he was cyanosed and his pulse was rapid. The epiglottis was exposed with the Jackson laryngeal speculum and several cuts made with a sharp knife; the epiglottis was not soft and gray as in edema, but hard, red and indurated. His pulse became weaker, and he stopped breathing. Artificial respiration with oxygen administered through a tube below the swollen epiglottis restored breathing. A rapid tracheotomy, under local anesthesia was performed, and a No. 2 tracheotomy tube introduced. The wound packed with iodoform gauze, and no stitches were taken.

As soon as the trachea was opened his breathing became better, and after oxygen was kept up for a few minutes through a rubber catheter along the tube, his color improved.

The secretion through the tube was a bloody, thin mucopus for the first few hours, then it became thicker and more tenacious; the tube was rapidly blocked with this thick secretion and the inner tube had to be changed every few minutes for the first 48 hours. On the third day it was possible to slip in a larger tube; but the inner tube still had to be changed every 10 or 15 minutes until the fifth day, when it could go for a few hours.

The epiglottis on the second day was still thick and swollen and covered with a thick, gummy secretion. It gradually diminished in size, and on the fifth day the swelling almost entirely disappeared, and offered no further obstruction to breathing. On the sixth day the tracheotomy tube was removed.

The temperature immediately after the operation was 104,° pulse 136, respiration 36, and at 6 p. m. the temperature went to 104.6.° For five days it ranged between 101.6° to 103.6.° It was normal on the seventh day and from then on. On the second day he developed pericarditis and pleurisy. For the first two days he suffered from abdominal distention and was expelling gas.

The urinalysis when he entered was negative. The next day he had a great deal of albumin and casts in the urine; the urine was normal again on the sixth day.

His blood count on admission was 22,000 white cells; with 85 per cent leucocytes. On the fourth day it was 13,000 white cells, and 65 per cent leucocytes.

Repeated cultures were negative for diphtheria; the first culture taken from the surface of the epiglottis after it was incised contained staphylococci and streptococci. Subsequent cultures from the

trachea contained only staphylococci. At one time the pathologist reported that he isolated two distinct types of organisms, 1 gm. positive, and the other negative.

He left the hospital on the tenth day and the wound was closed on the seventeenth day. At no time were the glands of the neck involved.

The most striking points about this case are the suddenness of onset and the rapidity with which symptoms reached an alarming stage and almost a fatal termination.

In review of the literature, I found only one case similar to this one, and that one was reported by Thiesen, of Albany. Whether these cases are really so rare, or whether many die undiagnosed is difficult to tell. It is, however, a complication which, on account of its gravity should be kept in mind in every case of acute respiratory infection.

While some may hold that this disease is a distinct pathological entity, I think that Semon is right in upholding the German school which contends that all infections of the air passages represent merely varying degrees of virulence of the same process. Why some acute respiratory infections are more limited than others in the extent of tissue involved and the degree of the virulence of the infection, is as yet undetermined; fortunately, acute respiratory infections, which are so common, are but rarely complicated with this dangerous and probably often fatal involvement of the epiglottis, and yet in any case of acute respiratory infection it is impossible to fortell this complication, which requires immediate relief to safe the life of the patient. It is a possibility which should be kept in mind at the first suggestion of dyspnea, when a rapid tracheotomy may prove a life-saving procedure, and a short delay might be fatal.

In doing the tracheotomy, I excised an oval piece of the anterior tracheal wall, as advocated by Hajek. I believe that this facilitated the reintroduction of the outer tube, which in this case had to be changed very frequently, and assisted in the expulsion of the thick, tenacious mucus which the child expelled while the tubes were being changed. At times he expelled large pieces that resembled diphtheritic membrane.

The general care of the patient was in the hands of Dr. N. N. Allen, the pediatrist.

647 Kress Building.

FOREIGN BODIES IN ESOPHAGUS WITH RESPIRATORY SYMPTOMS COMPLICATING DIAGNOSIS: REPORT OF FOUR CASES.

DR. J. B. NAFTZGER and DR. T. R. GITTINS, Sioux City, Iowa.

The following case reports are presented for two reasons:

1. As esophageal foreign body cases, they are of some interest.

2. As esophageal foreign body cases wherein respiratory symptoms were prominent, they are possibly of greater interest.

Case 1: Metallic Foreign Body in the Esophagus Eight Months.

Name: W. O. P., age 3 years.

History: In May, 1919, child began to have difficulty in swallowing coarse food, especially meat. There seemed to be no trouble with soft foods or liquids at this time. This difficulty continued for several months, during which time the child was losing weight and strength. There was gradually more difficulty in swallowing food. In October, about six months after the beginning of the trouble, the tonsils and adenoids were removed. The parents were told that this would relieve the difficulty in swallowing and breathing. Soon after this operation the patient began to cough. There was considerable wheezing respiration and dyspnea. At times the wheezing respiration would become very marked and there would be a rattling noise in the throat. No cyanosis. A doctor was called at this time and treated the patient for asthma and bronchitis. There was increasing difficulty in swallowing even soft foods now. At times some of the food would be regurgitated. About two weeks ago patient became quite ill. There was a high fever, sore throat, much cough and dyspnea. There has been a rapid loss of weight and strength. The day before this patient came under our observation she was seen by Dr. Coe, of Wakefield, Neb., who X-rayed the chest and discovered the metallic foreign body in the esophagus.

Examination: Child of 3 years, very emaciated. Marked pallor of the skin, rapid wheezing respiration, some cough. Throat: Tonsils have been removed. Upper end of the esophagus shows much swelling and ulceration. The larynx is pushed forward by the swollen esophagus and there is some edema of the epiglottis and over both arytenoids. There is a dirty gray membrane over the mucosa at the upper end of the esophagus, which bleeds very easily. Chest: Good expansion each side, breath sounds heard well over

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication Dec. 16, 1925.

each lung, numerous moist rales everywhere. X-ray of the chest shows a metallic foreign body in the upper portion of the esophagus, apparently 1¼x¾ inches. Temperature: 101.4.° Blood count: 22,000. Urinalysis: Negative.

Treatment: Patient was anesthetized with ether and the upper end of the esophagus was examined with the laryngoscope (Jackson). There was very marked swelling and ulceration of the mucosa and there was free bleeding. A small Jackson esophagoscope was passed carefully through this ulcerated and swollen area until the foreign body was seen about 1½ inches below the upper end of the

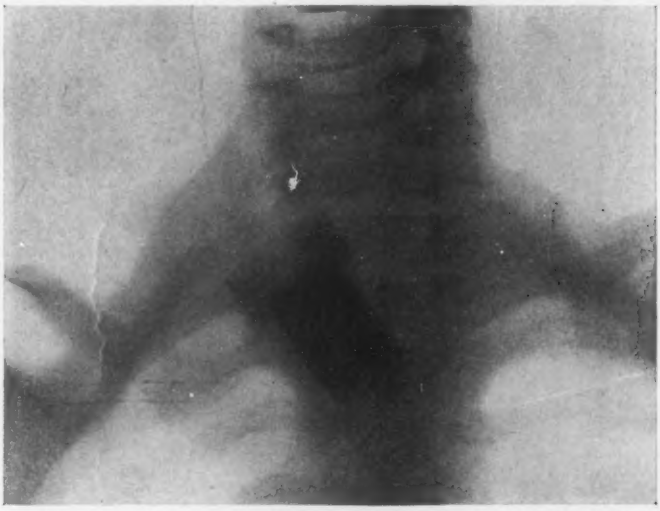

Fig. 1. Case 1.

esophagus. It was very firmly embedded in the tissues and took considerable force to dislodge. It was slowly and carefully moved from side to side, up and down, to gradually loosen it from the surrounding tissues. There was free bleeding when the foreign body was removed. The patient was returned to bed after fifteen minutes work in apparently good condition. Fluids were given as soon as the child was conscious and she seemed to swallow quite well. The first two days she had liquids only, but the third day some soft food was given. There was very rapid improvement in her general condition. The cough and dyspnea disappeared rapidly. When the patient left the hospital the parents were instructed to

return with her in two weeks time because of the possibility of a stricture. However, the patient did not return until Mar. 18, 1920, practically three months after the foreign body was removed. The child had eaten some roast beef the day before and had been unable to swallow anything since. Upper esophagoscopy without anesthesia was done and after removing some of the roast beef a stricture of the esophagus was encountered about 1½ to 2 inches from the upper opening of the esophagus. The stricture was dilated every day for four or five days and the patient again returned home, with the instruction to return in one month. The stricture has been dilated at intervals during the past few years and at present patient is eating practically all kinds of food.

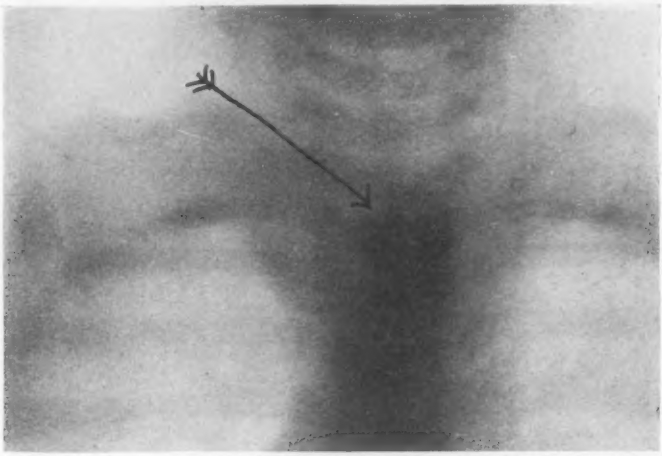

Fig. 2. Case 2.

Case 2: I. M., age 3 years. Mar. 24, 1922.

History: On Jan. 28, practically two months ago, child had severe choking spell, during which spell there was much cough and some cyanosis. Has been unable to swallow solid food since. During the past few weeks respiratory symptoms have been more pronounced. There has been cough, laryngeal stridor, and hoarse cry with rapid labored respirations. Child has been able to swallow liquids only. No history of foreign body aspiration.

Examination: Very emaciated restless child of about 3 years, marked dyspnea and hoarse cry and some cyanosis. Temperature, 99.2.° Pulse, 120. W. B. C., 12,800. Expansion quite good each

chest, breath sounds heard over all each lung, but many moist rales. Tonsils of moderate size and chronically infected. Some adenoid tissue.

X-ray chest: Foreign body, apparently a button, in the upper portion of the esophagus.

Treatment: Pharynx and larynx examined with child-size Jackson laryngoscope. Esophagoscope passed. Foreign body observed at the first constriction of the esophagus. Mucosa of esophagus at this point badly swollen and necrotic. Larynx and upper portion or trachea were pushed forward by the inflammation of the esophageal tissues. Breathing was very labored during the exposure and

Fig. 3. Case 3.

removal of the button. Some bleeding and sloughing of necrotic tissues, as foreign body was removed. Patient was dismissed on the second day. Respiratory difficulty practically entirely disappeared. There was some difficulty in swallowing, but soft food was taken easily. Patient was advised to return in a few weeks for observation, because it was felt that a stricture of the esophagus would develop. However, patient has not been seen since leaving hospital.

Case 3: Name: Baby W. W., age 3 months. Apr. 12, 1918.

History: Three days ago child became suddenly sick with cough and difficulty in breathing and a croupy cry. Parents and family doctor felt that the child was suffering with laryngeal diphtheria. Has had considerable difficulty in swallowing. Symptoms have be-

come worse during past twenty-four hours. Child will not swallow anything. Has a very hoarse cry. Rapid shallow respirations and some cyanosis. Temperature, 103.°

Examination: Chest: Breath sounds loud each side. Some moist rales. Expansion good each chest. Pharynx and tonsils negative.

X-rays: Large screw in upper portion esophagus.

Treatment: Examination with child-size Jackson laryngoscope. No anesthetic. Large screw located and quite easily removed from the upper end of the esophagus. There was considerable pressure forward against the larynx and trachea by the foreign body. Re-

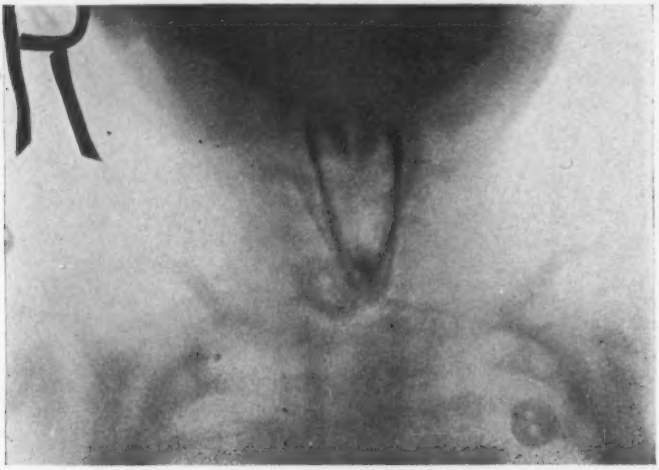

Fig. 4. Case 4. frontal view.

covery was rapid and child was dismissed next day with no respiratory symptoms.

Case 4: Name: Alicia T., age 4 weeks. Nov. 17, 1924.

History: At 9:30 this morning, five hours ago, mother left baby in room for a few minutes with a sister, age 1½ years. Mother returned. Baby was choking and quite cyanotic. Some cough and rapid breathing. Mother noticed that two safety pins had disappeared. Dyspnea was severe at times while patient was being brought to hospital.

Examination: Baby very restless. Hoarse cry. Rapid respirations. Temperature, 98.8.° W. B. C., 15,300. Breath sounds loud

and clear each lung. Expansion good each chest. Some moist rales. No cyanosis.

X-rays: Two open safety pins, points up, in the upper esophagus.

Treatment: Patient examined with child-size Jackson laryngoscope. No anesthetic. Point of one pin removed from where it entered the tissues of the upper esophagus. Pin rotated with point protected and removed. Same procedure with the other safety pin. One pin was pointing to the right, and one to the left. Time of operation, fifteen minutes. Patient in good condition on leaving table.

Fig. 5. Case 4, lateral view.

Nov. 20, 1924: No difficulty in breathing or swallowing. Dismissed in good condition.

REMARKS.

Case 1: Patient was treated for stomach trouble first. Later diagnosis of asthma and bronchitis was made because of the respiratory symptoms. The tonsils and adenoids were removed, supposedly to relieve the respiratory symptoms and difficulty in swallowing. After eight months of treatment under three or four doctors in three different states, an X-ray of the chest was finally taken, which revealed the metal foreign body in the esophagus.

Case 2. For two months there had been difficulty in swallowing. During the last two weeks of the illness respiratory symptoms had been very pronounced. Several days before the foreign body was

removed, there was marked dyspnea and some cyanosis and cough. X-ray revealed button in the upper portion of the esophagus. Its removal was followed by immediate relief of the respiratory symptoms and great improvement in swallowing.

Case 3: Baby, age 3 months, entered the hospital with a diagnosis of laryngeal diphtheria. There were two cases of diphtheria in the family at the time that the baby developed laryngeal stridor. The lack of fever or evidence of toxemia prompted us to take an X-ray of the chest, which revealed a large screw in the upper portion of the esophagus. Removal was followed rapidly by cessation of respiratory symptoms.

Case 4: Two open safety pins in the upper portion of the esophagus in a child, age 4 weeks, gave rise to rather severe respiratory symptoms. In this case the diagnosis was made early because the mother insisted that two safety pins had disappeared suddenly. Without such a history the respiratory symptoms would have been very misleading. X-ray revealed two safety pins, open and points up, in the upper end of esophagus.

A CORRECTION.

On Page 223, THE LARYNGOSCOPE, March, 1926, a discussion of Dr. Clerf's Paper, "Foreign Bodies in the Tracheobronchial Tree," was credited to Dr. Vaughan, through an error. This discussion should have been credited to Dr. T. W. Moore, of Huntington, West Virginia.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, November 25, 1925.

(*Continued from page 312.*)

Stenosis of the Pharynx. Dr. Sidney Yankauer.

This patient had a tonsillectomy done about four years ago. Following the operation stenosis of the pharynx took place, the soft palate being adherent to the posterior pharyngeal wall over the greater portion of its extent. She was operated upon two or three times by different surgeons without successful outcome, and two years ago presented herself to me with a complete adhesion of the mucosa of the soft palate to the posterior pharyngeal wall, with the exception of a small opening in the center, through which a probe could be passed. The operation which I performed on her at that time will be described later.

The result of the operation was somewhat impaired by secondary hemorrhage which occurred shortly after operating on the right side, so that at the present time there is more induration on the right side than on the left, but the opening remains patent and the patient is able to breathe through her nose with comfort. It is necessary for her to maintain the patency of the pharynx by the use of the dilator which is here presented. The patient uses this dilator herself and will demonstrate the method of its use.

Treatment of Some of the Unpleasant Sequelae of Tonsillectomy. Dr. Sidney Yankauer.

DISCUSSION.

DR. I. M. HELLER: What Dr. Yankauer said about the anterior and posterior pillars we have all seen; and yet I have seen as perfect an operation as I was able to make followed later by adhesions, although there were no symptoms. Again, when I was not so well pleased with an operation, I was surprised to see the pillar stand out quite distinctly, yet these patients often made more complaint. I have seen these same conditions occur with men who are the best operators in New York City, and I don't know how these adhesions can be prevented. If Dr. Yankauer will tell us, we will be very grateful.

DR. ROBERT H. FOWLER: Will Dr. Yankauer tell us the *time* when he uses this massage after the operation? In my experience, massage and *stretching the scar* during the first month after operation have helped and, as a matter of fact, most of the patients show the scar loosening up six months later, so that thereafter it is better than might be expected. It will be a pleasure to try out the more elaborate massage described by Dr. Yankauer, and I am sure that his suggestions will be very valuable.

DR. HURD: I don't think I heard Dr. Yankauer clearly. He spoke of an extensive dissection down so as to anchor the sides of the fossa. I have seen this where the massage has done a lot of good. They come complaining of the tongue being bound to the throat, and after they are shown how to massage between the tongue and fossa, after a while that area is stretched out and they get over the sensation. Another thing: there are some persons who have a tight nasopharynx—have a chronic constriction there—and putting the finger into the nasopharynx and stretching the muscle does a lot of good.

DR. L. D. ALEXANDER: This is a most valuable contribution. For a number of years I have maintained that it is good technic in singers to leave the lingual plica, trimming it if necessary. Dr. Yankauer has introduced something new and of great interest. I hope he will elaborate it so that we can relieve some of these distressing cases.

DR. JOHN HORN: It has been a great pleasure to me to hear Dr. Yankauer's paper, and I am greatly interested in his suggestions in regard to massage. I have done a great many tonsillectomies in my day, but I don't know of any of my cases that have needed massage. I feel sure, however, that most, if not all, of them are due to traumatism.

DR. YANKAUER: A number of operators in describing the operation speak of the capsule having two layers. If you will examine the capsule carefully in doing the dissection operation you will find that it consists of a dense layer of fibrous tissue, thick, hard membrane closely associated with the tonsil, and on the surface of the capsule is a layer of thin, loose areolar tissue, that normally separates the capsule of the tonsil from the fascia of the pharynx; if you leave that in the throat, your pillars will not become adherent. You have to scrape the capsule clean before you can put on the snare, etc., and then you will have no adhesions; but if your tonsillectomy is not done with such care that you take away the loose areolar tissue and take away the fascia of the pharynx, you will always have adhesions between the pillars. These adhesions are sometimes very annoying to the patient.

As to the time of beginning the massage, most of the cases that I have had have come to me a long while after the tonsillectomy. The last patient was tonsillectomized over a year ago, and she had so much irritation that she had to consult a laryngologist. She had been treated by X-rays and all sorts of things for a year without any relief. The first massage cleared away the symptoms for twenty-four hours, and after the treatment she was promptly relieved. The massage, coming a long while after the tonsillar operation, gives results which are almost immediate. The patient will tell you after the first treatment that the throat feels better. This massage is done by my nurse, for she has learned to do them quite skillfully and without gagging the patients too much, and anyone who has had any experience and has a gentle touch, and knows the anatomy of the throat can perform this quite easily, and the benefit to the patient is immediate and the results most satisfactory.

The question was brought up as to whether I was producing a psychic effect on these patients, but I am sure it is nothing of the kind. The patient has been informed of the need to have the throat massaged, and it was done. I do not usually see them until after the third or fourth treatment, and they come back and describe the improvement, and so far it has not failed to relieve the symptoms where there were not neuroses but due to the scars. Such a case as was described by Dr. Hurd I see once in a while, and that is the manipulation we use—press the tongue down and massage the scar, just as in any other massage of scars.

Technic of Local Anesthesia for Tonsillectomy. Dr. Sidney Yankauer and Dr. Rudolph Kramer.

In 1909* one of us (Y) advocated blocking of the middle and posterior palatine nerves at their exit from the foramina in the hard palate, by the injection of novocain. This procedure, in addition to the usual surface swabbing with cocain and the pillar injection of novocain, satisfactorily anesthetized the tonsillar region in a large number of cases; but in many, pain was felt on snaring off the tonsil. After further study of the question, we found a method of local anesthesia which has proved entirely satisfactory.

The tonsils are innervated by the middle and posterior palatine nerves and by branches of the glossopharyngeal nerve. The posterior palatine nerves emerge from the accessory posterior palatine canals, the middle palatine nerves from the external canals, anterior and mesial to the hamular process, and pass downwards and backwards to the upper pole of the tonsil. The glossopharyngeal nerves send branches through the pharyngeal aponeurosis to the tonsillar fossa where a plexus is formed which ultimately innervates the tonsil.

The procedure employed is based upon the blocking of the middle and posterior palatine nerves and the tonsillar branches of the glossopharyngeus. A subcutaneous injection of morphin sulphate gr. ¼ and atropin sulphate gr. 1/150 is given one-half hour before the operation. The mucosa of the

*The Laryngoscope, May, 1909. Tonsillectomy. S. Yankauer.

hard palate, anterior to the tip of the hamular process, and the anterior and posterior palatine arches are swabbed with a solution of cocain hyrdochlorid 20 per cent and adrenalin hydrochlorid (1:6000). Two applications at intervals of three minutes are all that is usually required for this step. The palatine nerves are then blocked by a solution of novocain hydrochlorid 2 per cent and adrenalin hydrochlorid solution (1:50,000). This is injected by means of a straight tonsil needle 10 c.m. long, the distal centimeter having a diameter of 1 m.m. and the point beveled at an angle of 45.° Luer syringe has been found most satisfactory for injection.

To anesthetize the middle and posterior palatine nerves, the needle penetrates the mucosa of the hard palate just anterior and mesial to the base of the hamular process. The barrel of the syringe is in the angle of the mouth, on the side opposite to the point of injection. The needle is advanced until the hard palate is reached and then the solution is injected until a wheel appears. This requires about 1 c.c. of solution.

The glossopharyngeal branches are infiltrated in four places. The solution used is novocain hydrochlorid 1 per cent. The first point of injection is immediately posterior to the tonsil at the level of the tip of the lower pole. The second point is in the depths of the fossa between the plica triangularis and the anterior pillar as near the lower pole as possible. As we have been unable to find any name attached to this fossa, we have called it the fossa triangularis. About 0.5 c.c. of solution is injected in each of these regions. The third injection is made at the junction of the anterior pillar and the tongue so that a wheal is raised on the dorsum of the tongue. 0.25 to 0.5 c.c. of solution suffices for this.

The fourth injection is made through the anterior pillar at the level of the equator of the tonsil. The needle hugs the external surface of the tonsillar capsule until the narrow distal centimeter of the needle has been buried in the tissues. About 2 c.c. of solution is injected here, with the result that the tonsil is pushed, to a slight extent, towards the median line. The operation can be proceeded with, two to three minutes after completion of infiltration of the second tonsil. Adrenalin is not added to the novocain hydrochlorid 1 per cent injected about the tonsillar bed, because we fear that the vasoconstrictor action of the adrenalin may temporarily close bleeding points which may lead to secondary hemorrhage when the delayed vasodilatation of adrenalin sets in. We have found the "hooked" tongue depressor of Yankauer very useful in exposing the fossae in front of and behind the lower pole of the tonsil.

All the tonsillar bed injections are extracapsular. The tonsil and its infected crypts are not entered. The necessity for the four points of infiltration about the tonsil results from the peculiarities of the course of the glossopharyngeus. The nerve trunk is external to the tonsil and sends branches to the tonsil which perforate the capsule. These branches are infiltrated by the injection of the anterior pillar at the level of the middle of the tonsil. As the glossopharyngeus curves forward to the tongue, below the tonsil, it sends off, as a rule, one branch which ascends posterior to the lower pole of the tonsil, and another which ascends in front of the tonsil, passing through the depths of the fossa triangularis. These two branches are blocked by the injection anterior and posterior to the lower pole of the tonsil. The junction of the anterior pillar and the base of the tongue is infiltrated so as to prevent the pain due to the cutting and pulling of the snare acting on the attachment of the anterior pillar as it passes on to the tongue.

Where extensive and dense peritonsillar scars, the residue of former abscesses, exist the snaring off of the tonsil may be the cause of momentary pain. The cause of the pain and the reason for failure of complete anesthesia in these cases is not definite. We feel that a spasm of the superior constrictor muscle as the snare pulls on the pedicle of the tonsil is the probable cause of the pain. The scarring of the peritonsillar tissues prevents the infiltration of the novocain solution around branches of the glossopharyngeal nerves. An additional factor may be the distortion of the course of the nerves due to the scarring.

DISCUSSION.

DR. JAMES J. KING: I regret that I must condemn Dr. Kramer's technic as unsafe. I have done thousands of tonsillectomies under local anesthesia in patients between the ages of 15 and 80 years of age. Local anesthesia is the method of choice in my hands.

We all should know that there is greater danger from toxicity when cocain is used in the combination with novocain. I cannot condemn too strongly his procedure of painting the pharynx with 20 per cent cocain solution and the injecting novocain. This use of cocain in tonsillectomy may have been justified prior to 1905, before the discovery of novocain, a safe and satisfactory local anesthetic, but I can see no justification for such a dangerous procedure in 1925. If it were necessary there might be some excuse for its use. The combination is condemned by committee of the A.M.A. on the study of local anesthetics. In 1923 I published a paper on Tonsillectomy in Adults Under Local Anesthesia, in which a safe and satisfactory technic is advocated. There have been many deaths reported from the combined use of cocain and novocain. The doctor should learn a safe technic with novocain anesthesia.

In my technic a preliminary sedative is given to the patient. Then by the use of a fine needle (25 or 26 gauge) 0.5 per cent novocain is infiltrated around the tonsils.

This produces a very efficient anesthesia for the operation and its safety recommends it above all others so far as our knowledge of today goes.

DR. L. D. ALEXANDER: I had the pleasure of seeing Dr. Braun do some of this work. He has found that by using one-tenth of 1 per cent cocain with air pressure of 10 to 12 pounds he gets as much anesthesia as with 10 per cent. Certainly, if we are going to use cocain, we should try that rather than use a strong solution.

DR. JOHN HORN: I merely wish to give a warning to those present against the use of 20 per cent cocain solution, applied as suggested by the reader of the paper.

DR. HURD: I don't know why they put any local anesthesia on at all without preliminary swabbing or any other anesthesia. Make one or two injections, and let the needle go. They don't gag any more, if you do or don't. I have much doubt about how much anesthesia you want. I have used novocain on one side and used normal salin on the other side, and the patient said that it did not hurt on either side; on the other hand, I have heard them yell with all the cocain you could put in.

DR. YANKAUER: It is not strange that we get a good deal more poison from cocain by using a very weak solution than a strong solution, for the strong solution . . . The amount of cocain left in the patient is exceedingly small. There is not more than . . . left in the spot, and that is brought out of the throat again when it is swabbed. There is more left in the throat by any spray of 0.5 per cent solution than if you take a swab and touch the . . with it at the side. We have done many tonsillectomies in that way and have yet to see anything that could resemble either cocain or novocain poisoning.

DR. R. KRAMER: Dr. Hurd's remarks bring up the whole question of local anesthesia as opposed to general anesthesia, and I did not want to take that up in this paper. I have seen about 200 cases of lung abscess, one-fourth of which followed tonsillectomy under general anesthesia, and when you see that you are not so keen on general anesthesia. I have never seen one following local anesthesia.

DR. HELLER: What percentage in adults?

DR. KRAMER: The greater percentage was in adults—some occurred in children, but the great percentage was in adults. They have been reported following tonsillectomy under local anesthesia. I suspect that in these cases the larynx was anesthetized as well as the pharynx, and they had an aspiration infection. Of course, there must be a certain number of embolic lung abscesses, but most of you will agree that this is small compared with aspiration. Having seen these cases and feeling very strongly about using any general anesthetic in any upper respiratory tract operation, if I can possibly get away with it, I do it under local anesthesia. If you don't use cocain in

the throat, you get pain when you stick the needle in. You can get the patient to stand this pain, but the reason for getting complete anesthesia is to popularize the method of doing it under local anesthesia. The patient does not object to local anesthesia, it is usually the doctor. In order to get the method popularized and accepted, you must have a painless operation. If you don't, the patient will report it and the next patient will not permit local anesthesia.

DR. FRANCIS WHITE: The subject has been covered thoroughly by Dr. Yankauer's paper and I hope he will have the opportunity of presenting some of his patients later. The subject is very interesting. I believe, however, we see fewer than formerly. We used to see many that had some pretty bad scars; but with the improved facilities and technic that are now in common use, we do not see these horrible results so frequently.

Dr. Hurd brought up the point of injecting normal salin into the tonsil and getting a fairly painless tonsillectomy. Of course, much of that may have been psychic but it is conceivable, and I think some have tried the injection of normal salin solution. If you reach the nerve, you get a pretty good anesthesia. That may be the factor in a case of this kind.

With reference to the strength of the cocain applied, I suppose it is a question of personal experience and personal feeling. I have never used anything but 20 per cent cocain with adrenalin for operative work. I have never seen a case of cocain fatality. I have seen one case of cocain poisoning that almost ended fatally. This was due to the fact that cocain was injected by mistake instead of novocain. I think this is what has happened in other cases of fatalities following local anesthesia. The surgeon thought he injected novocain and, instead, he used cocain. The resulting fatality is ascribed to surface application of cocain and the injection of novocain, whereas it is purely a novocain injection poisoning. The same accident has happened to general surgeons and to urologists. In my experience, a 20 per cent solution of cocain hydrochlorid with adrenalin hydrochlorid, if applied to the surface of the mucosa, does not result in poisoning.

SECTION ON OTOLOGY.

December 11, 1925.

Case of Chronic Otitis Media Purulenta, Mastoiditis, Cholesteatoma, Meningitis Sympathica, Perisinus Abscess, Abscess in Posterior Cranial Fossa. Dr. Mark J. Gottlieb.

This case is reported for the reason that an operation was undertaken on her right temporal bone with the idea of doing a radical mastoid operation and labyrinthectomy because she was developing meningitis. The right labyrinth did not respond to caloric stimulation, while the left was found to be active. It was presumed before the operation that she had a latent labyrinthitis and the beginning meningitis which was due to an extension from a chronic suppuration of the labyrinth. However, after the mastoid cortex was removed, such conditions were found as to make it necessary to revise the pre-operation program.

G. S., age 15 years, admitted on Apr. 1, 1925.

Chief Complaint: Discharge from both ears, chills and fever.

Past History: She had diphtheria at the age of 7, since that time has had discharge from both ears and impaired hearing. The discharge ceases at times, and then recurs.

Present History: One week before admission, the discharge from both ears ceased. The patient then experienced pain on the right side of her neck and says that her neck became swollen. This was accompanied by chills and fever. The swelling in the neck lasted two days. Had not complained of headache or mastoid tenderness.

On the day of admission, the left eardrum showed an old perforation situated high up in the anterior portion of the drum. The posterior canal wall was thickened and was deep in color, but not acutely inflamed. The canal contained some cerumen or inspissated discharge. The right eardrum had a perforation in the upper posterior quadrant, a thick, foul discharge issued from the perforation. The posterior superior portion of the canal sagged moderately and the posterior canal was acutely inflamed. No edema, redness or tenderness was found over either mastoid processes. The patient was mentally alert and did not complain of headache or other symptoms which might have left one to suppose that a serious process was going on in the patient's head. The reflexes were apparently normal. No neck rigidity or Kernig. No observation was made regarding the presence or absence of spontaneous nystagmus at the time of admission. The temperature was 97.2° and the pulse 116. The blood count on the following day was: leucocytes 19,200, polys 68 per cent, and mononuclears 32 per cent.

The patient continued to be very comfortable with only slight rises of temperature until four days after admission, when the service was notified that she had become stuporous and drowsy, complaining of headache and had some neck rigidity. A spinal puncture done at this time was productive of a fluid under pressure and turbid. The result of the examination of this fluid was reported on the following day as follows: leucocytes 1260 per c.m.m., polys 97 per cent. Culture and smear were negative. The temperature gradually rose to 102° at the beginning of the meningeal symptoms and then ascended to 104.6.° Examination at this time revealed a spontaneous horizontal nystagmus to the left, occasional fine horizontal nystagmus to the right, right ear discharging foul smelling pus, fullness in the posterior canal wall near region of the drum. Left ear showed no change from that found on the first examination. Internal squint of right eye, moderate neck rigidity, Kernig is suggestive on the left side only. Unsustained ankle clonus on the right side. Babinski present on both sides. The right labyrinth does not respond to douching with water at 68,° while the left labyrinth was found to be active.

Preoperative Diagnosis: Right chronic mastoiditis, dangerous type; latent labyrinthitis-meningitis.

Operative Program: Immediate radical mastoidectomy, drainage of labyrinth by Hinsberg method.

Findings at Operation: Cortex of mastoid was thick. When uncapped, a large quantity of thick, foul smelling pus exuded. The sinus was found exposed throughout its entire extent. It was covered with large, thick granulations. The sinus abutted against the posterior canal wall and filled in the mastoid antrum, obstructing the approach to the tympanum by the aditus route. An effort was made to open the tympanum through the zygomatic root and a small epidural abscess was found in this location. While endeavoring to take down the posterior canal wall, the anesthetist noticed a twitching of the face. A trickle of thin, cloudy fluid was then noticed just above the knee of the sinus. An anatomical forceps was inserted above the sinus for about 1½ inches, and a large abscess of foul smelling, thin liquid mixed with pus and white, oily flakes was evacuated. The forceps actually fell into the cavity. The evacuation was produced by the spreading apart of the forceps. The bottom of the cavity was not felt. An iodoform gauze drain was placed in this cavity and the operation was terminated without suturing the wound.

Within a few hours, the patient again became conscious and remained comfortable until three days later, when she became unconscious, respiration became very slow, developed pulmonary edema, and then died. After the operation it was noticed that she had an incomplete paralysis of the right side of the face.

The writer hopes that this presentation will evoke discussion on the following points:

Was the labyrinth actually the cause of the beginning meningitis, or did the process in the mastoid extend by necrosis into the posterior fossa, dissect up the dura from the posterior surface of the petrous portion of the temporal bone and surround and impinge upon the auditory nerve to such extent as to block impulses coming from the end organ?

Was the collection of pus in the posterior fossa epidural or subdural?

Was the collection of pus in the substance of the cerebellum?

How is it possible to account for the internal strabismus of the right eye?

Would one be justified in obliterating the lateral sinus to facilitate drainage, situated as it was in this case, acting as a partial barrier to the free discharge of pus from the posterior fossa?

DISCUSSION.

DR. NOAH SCHILLER said that chronic otorrhea is always a serious matter, especially when accompanied by meningeal complications. He then told of a man age 38 years, who came under observation three months ago, with a history of otorrhea of five months' duration, and complaining of pain out of all proportion to what could be accounted for, and an increase of the discharge within the past three days. He was admitted to the hospital and operation revealed a temporosphenoidal abscess. Spinal puncture showed pus cells and pneumococci. Here was a case of only five months' duration, with an abscess of the brain. At the same time, many cases of chronic otorrhea can be treated palliatively. One patient, a boy age 10 years, had a canal filled with polypi. These were removed by pure caustic silver nitrate. The treatment was continued day after day for three or four weeks, the granulations were destroyed, and a result obtained equaling that from a radical operation, without the attendant dangers. It is very difficult, however, to make a diagnosis before symptoms of intracranial complications develop, to decide whether one should do a radical operation or employ palliative treatment. Dr. Gottlieb's case showed meningeal symptoms and required operation, etc.

DR. H. M. SCHEER said that the subject of chronic otitis media is one of every day occurrence and all know the seriousness with which it should be considered. It should be watched cautiously and taken great care of. It is the duty of each member of the Section as well as other otologists to get out more frequently and impress on the general practitioner that intracranial complications to O.M.P.C. are of more frequent occurrence than they realize. Such cases as the one under discussion and those mentioned by Dr. Schiller are seen every week at any of the larger otological institutions. Within the last three weeks at the Manhattan Eye, Ear and Throat Hospital, in the service of Drs. J. R. Page and J. D. Richards, two such cases occurred. One patient came in with a temperature of 102° and symptoms of meningitis. Both ears had been discharging, and one of them appeared to be in an acute condition. Spinal puncture showed cloudy fluid, but no bacteria. Dr. Richards operated on the mastoid, but nothing much was found in the bone. As the operation continued a big sinus leading into the brain was exposed, in necrotic dura, in the temporosphenoidal region and an ounce of pus was evacuated. Dr. Richards did a petrous pyramid operation. The patient lived for ten days and was perfectly conscious, but died at the end of this time. Another case operated upon recently at the Manhattan Hospital, a young man was admitted to the service at night with a temperature of 102,° rigid with meningitis. Spinal culture gave fluid under pressure, with no bacteria. His labyrinth was active. The day of admission he was operated upon, and the mastoid was found to be sclerotic; the only point that seemed suspicious of pathology was a necrotic area at tegmen antri. The dura was opened up (a portion near the surface of the temporal bone was normal), but as more was exposed, pus exuded between the brain and the dural plate; further exposure revealed a sinus that led to the temporosphenoidal lobe and there was another brain abscess. Anyone associated

with the special institutions could cite instances showing how frequently these occur.

DR. GOTTLIEB, replying to Dr. Schiller, said that in the presence of a beginning meningitis or a meningitis sympathica and not finding anything else in the mastoid, he would be inclined to open the labyrinth every time, if the labyrinth were dead. Referring to the point brought out by Dr. Scheer, that one would have to see the operation, Dr. Gottlieb said that he had described the condition as accurately as he knew how.

The lateral sinus abutted against the posterior canal wall and practically filled in the mastoid antrum. The upper limit of the sinus obstructed satisfactory drainage of the large abscess in the posterior fossa. It occurred to us that obliteration of the sinus would be helpful, but we were confronted with the following conditions: it was found impractical to tie off the sinus, because to do this it would be necessary to bite off some of the posterior canal wall at its base. This procedure would have endangered the facial nerve. The only other way to obliterate the sinus is to pack a plug of gauze above and below. The sinus was covered by granulations. Injury to the sinus wall may have invited a phlebitis which might have eventuated in a thrombosis extending into the jugular bulb and vein. For the above reasons, the sinus was not disturbed, but considering the outcome of the case it is our opinion now that it would have been wiser to block off the sinus.

The X-ray Diagnosis of the Diseases of the Petrous Pyramid. Dr. Harry A. Goalwin.

The petrous portion of the temporal bone is the most complicated structure of the most complicated part of the human skeleton—the skull. The complexity of its structure would alone be sufficient to explain the comparatively slow progress of aural Roentgenology. But there are still other reasons for the slow developments along the lines of the X-ray diagnosis of the diseases of the petrous portion of the temporal bone as compared with its mastoid portion:

1. In the mastoid portion pathological changes are demonstrable very early. In the presence of an acute otitis media, or even in inflammation of the external auditory canal, there is a rapid reduction of the air content of the mastoid cells which is demonstrable on the X-ray picture. In the pyramidal portion, on the other hand, the only early changes which are so demonstrable are fracture and tumor.

2. The authors who worked on the X-ray demonstration of the details of the petrous pyramid did so in the attempt of demonstrating *all* the details on *one* Roentgenogram, or two stereoscopic views. The engineer who wishes to study the simplest apparatus or instrument from its drawings, never attempts to do so from just one view. He takes two or three general views and a number of detail views and sections. Mayer first called attention to the impossibility of making a complete study of the petrous pyramid from one Roentgenogram and offered the view which he devised only as an additional aid to those of the workers who preceded him.

Even in the Roentgenography of the mastoid, which is widely used, the otologist does not realize the full benefit obtainable. When a diagnosis of mastoid involvement is made from the clinical and Roentgen findings there are certain anatomical details which the X-ray examination can give him that are of great help in the execution of the operative procedure he is to undertake. These details can only be obtained by a study of the petrous pyramid. They (these details) are: the distance of the sinus from the posterior wall of the external auditory canal, the distance of the dura from the roof of the canal, the thickness of the tegmen tympani and tegmen antri. These details are important not only for the operation, but as an indication for or against an operation, because they indicate the progress of the disease.

The complete X-ray examination of an ear requires four different views, each of which has a distinct and definite purpose. The first of these is the view of the mastoid you are all familiar with, which has been described by Sonnenkalb, Heidenhain, Law. The next is the lateral view of

the pyramid, which was described by Schüller. The third is the posteroanterior view of the pyramid taken at right angles to its axis, as described by de Kleyn and Stenvers. The fourth is a top view of the pyramid described by Mayer. In every case the opposite side should be studied for comparison.

We have simplified all these views in collaboration with Professor Schüller, of Vienna, so that, as in all our cranial Roentgenology, the central ray of the tube is always perpendicular to the plate. The manipulations necessary are therefore limited to the head.

Schüller determined by measurements that the line connecting the centers of the external and internal auditory meati of the same ear makes an angle of 30° with the horizontal plane of the head and remains within the vertical plane connecting the centers of the two external auditory meati. If we place therefore the patient's head on the plate so that the interaural plane is perpendicular to the plate and the horizontal plane makes an angle of 60° caudally with the plate, we can, by aiming at the external auditory meatus of the side which is on the plate, project the internal meatus within the external.

This Roentgenogram gives us a lateral view of the pyramid. As pointed out by Mayer, the distinctness and lack of distortion of this picture enables us to measure from it directly the location of the sigmoid sinus and of the attachment of the dura with respect to the external auditory canal, and the thickness of the tegmen tympani and tegmen antri. Our own practice is never to take direct measurements from the Roentgenograms as final, but to calculate the actual from the Roentgenographic measurements, making allowances for the focus-plate and object-plate distances, and for any angulation of the rays which strike the object. When the Roentgenogram is taken without the Bucky diaphragm such allowances are practically negligible in this particular Roentgenogram on account of the proximity of the parts to the plate. Since we use the Bucky diaphragm such calculations cannot be neglected.

In the adult skull the normal distance between the posterior wall of the external auditory canal and the anterior wall of the sinus is taken as 12 to 8 m.m. Anything below the minimum stated is taken as evidence of deepening of the sinus. The distance between the tegmen tympani and the roof of the external auditory canal is taken to be 4 to 5 m.m. in the normal adult skull.

The posteroanterior view of the pyramid is obtained by placing the patient's head with the face toward the horizontal plate, the median plane making an angle of 45° with the plate on the side to be investigated, and the horizontal plane of the head making an angle of 10° with the plate (open cephalad). The central ray is pointed to the midpoint of the line connecting the external auditory meatus with the outer margin of the orbit of the side which is on the plate. On this view we see the semicircular canals, vestibule and internal auditory meatus, in addition to the mastoid process and the upper and lower outlines of the pyramids. Changes in the structures mentioned are easily demonstrated on this Roentgenogram. Fractures may be seen in this view to run from the jugular fossa across the pyramid, or they may run parallel with the pyramidal axis, through the internal meatus.

The top X-ray view of the pyramid was originally described by Mayer. He obtains it by placing the head, face up and to one side, with the side to be investigated on the plate which makes an angle of 45° with the horizontal table, and aiming the central ray toward the external meatus of the side to be investigated. We place the head in the same position, but keep the plate horizontal. The object of this Roentgenogram is, to quote from Mayer's original communication, "To demonstrate:

1. The external auditory canal throughout its extent.
2. The lateral wall of the recessus epitympanicus.
3. The antrum.
4. The middle ear.

5. The ntire pneumatic system with the exception of the tip of the mastoid which is covered by the pyramidal shadow.

6. The sulcus sigmoideus."

The following cases will illustrate the clinical value of the various Roentgenograms described and the principles of their interpretations. On account of the difficulty of following these unfamiliar views we have grouped the cases not according to the pathological condition but according to the view on which the conditions have been demonstrated.

The first case shows a large cholesteatoma of the antrum with early breaking through of the tegmen antri. You will note that in the ordinary mastoid position one cannot tell definitely the relation of the defect to the external auditory meatus, while in the Schüller view this can be definitely measured.

The second case shows a normal, highly pneumatized right mastoid and pyramid and a markedly sclerosed left pyramid with a large left mastoid which shows atrophy of the cells. The attics are markedly enlarged on both sides, with atrophic, smooth walls. We are dealing primarily with bilateral tuberculosis of the attic. In the left ear, which is the only one that has a chronic suppurative otitis media, there has apparently occurred a secondary infection. This explains the atrophic mastoid associated with a sclerosed pyramid.

The next group of slides shows the pyramid in the posteroanterior, or Stenvers position.

The first slide shows the typical transverse fracture which extends from the jugular fossa upward and inward across the internal meatus.

The second slide shows an old fracture of the left pyramid which is parallel to the axis of the internal meatus. The internal auditory canal is enlarged and its walls thickened. Its lumen has a uniform diameter as contrasted with the enlargement produced by tumor, which is either irregular or spindle-shaped. This patient was struck by a falling ladder against a ship railing six years ago. Although he had definite signs of fracture at the time of the accident and now has marked reduction of hearing, two previous X-ray examinations failed to show any pathology. This view is the only one that can demonstrate this type of lesion.

The third slide shows erosion of the apices of both pyramids by a tumor of the middle fossa. The right pyramid shows also erosion of the superior semicircular canal and enlargement of the jugular fossa. The body of the sphenoid in this case is almost entirely destroyed, as you see in the next slide. The lesion has also extended into the frontal area and destroyed the roofs of both optic canals, as you see in the following slide. The patient had bilateral optic atrophy, dizziness and difficulty in maintaining her balance.

The next group of slides shows the pyramid in Mayer's top view. The first slide shows enlargement of the attic and aditus by cholesteatoma, so that the attic and antrum are merged into one common cavity.

The next slide shows bilateral hyperostosis and exostosis of the external auditory canals. Mayer has reported four similar cases. While pathology of the external auditory canal can usually be diagnosed without X-ray examination, there are cases where examination is of additional assistance. In atresia of the external meatus the X-ray examination will show whether the bony canal is closed or patent. In constriction or local swelling we can see whether we are dealing with overproduction of bone, or soft tissue, bone destruction by tumor, abscess, etc.

The last slide shows a punched out defect behind and below the left pyramid, just external to the jugular fossa, therefore immediately behind the stylomastoid foramen. The patient had all the symptoms of a tumor in this region, left facial paralysis, inability to maintain her equilibrium, etc., yet the only X-ray evidence of the tumor could be found in the view shown in this slide.

DISCUSSION.

DR. GEORGE S. DIXON said he was acquainted with the work Dr. Goalwin was doing and had been much interested in the cases shown. It would all be very well if all the bones were alike, but unfortunately for some of the conclusions drawn the petrous pyramid will have quite as many variations as the mastoid process. Examination of several hundreds of sections of the temporal bones had shown very few of them to be near alike. The capsule of the labyrinth varies greatly, the internal canal also, as well as the external; the antrum, the aditus, the attic are never the same. No two mastoids are ever the same in any individual or in any two individuals. It does not seem possible to find two bones exactly alike. X-ray diagnosis of disease of the semicircular canals seems to be drawing it rather fine, to say the least.

Dr. Goalwin had spoken of measuring the distance from the anterior of the sinus groove to the canal. Dr. Dixon said his method of measuring the location of the sinus is to use an angle of about 13° for the plate, which with the lateral angle of the face, will throw out the opposite side, and if the tube is properly placed and the ray directed at right angle to the table, the internal and external canals will be exactly superimposed. Experience has shown that it makes very little difference how far the canal seems to be from the anterior border of the sinus; it depends altogether upon the angle the sinus makes with Reid's base line; the nearer it comes to a right angle the closer it is to the canal, and if it is still increased the antrum cannot be reached without taking down the posterior canal wall. What Dr. Goalwin said about the tumor in the tip of the petrous apex was very interesting, and he was to be congratulated on obtaining such results.

DR. LAW did not think the members would be particularly interested in a discussion of the technique, but rather of what Dr. Goalwin in his diagnosis was able to detect. He was rather surprised, however, that Dr. Goalwin seemed to think he was the first to superimpose the canals, though the Doctor was certainly to be congratulated on his very remarkable interpretations. The pictures presented an excellent demonstration of the futility of writing an interpretation which can in turn be interpreted by the otologist. A consultation between the otologist and the Roentgenologist is most essential in arriving at a correct understanding of the condition, as could easily be seen by looking at these plates, particularly if one is not accustomed to studying them. It would be interesting to hear the opinion of any otologist present as to the practical value of the information gained in these different positions.

DR. MAYBAUM thought it a matter of considerable interest that in the past year two papers had been presented before this Section pertaining to the petrous bone, one relative to the surgery and now this one by Dr. Goalwin on the diagnosis by means of the X-ray of diseased conditions of the petrous bone. It is a curious fact that the otologist is no longer content with the problems and worries of every day practice of otology, but now seeks to invade fields which are still considered inaccessible.

The otologist would undoubtedly welcome any light which may be thrown upon the diagnosis of diseased conditions of the petrous portion. What practical value could be obtained from careful X-rays of this region? Take for example the question of diagnosis of acousticus tumors in obscure or early cases. Some years ago Cushing and others stated that by means of the X-ray an enlargement of the internal auditory meatus could be demonstrated on X-ray examination. Few otologists or neurologists have availed themselves of this finding. Does Dr. Goalwin consider it possible to obtain such findings by means of the X-ray?

In certain cases presenting the syndrome described by Gradenigo, in which there is extension through the petrous portion to its apex of an inflammatory condition, it would be of interest to determine the type of petrous bone with which we are dealing. In obscure cases it is of importance to determine whether we are dealing with a compact or a cellular structure. This brings out the question as to what relation the structure of the mastoid has to the structure of the petrous bone, i. e., whether in the cases of sclerotic mastoids

the result of a chronic suppurative process, a similar type of structure is found in the petrous bone. In mastoids of the infantile type, what are the structural findings in the petrous portion? Knowledge of the latter facts may have some bearing as to the pathway of infection in some cases of meningitis.

In the neurological service at Mt. Sinai Hospital there was an interesting case a year ago, in which the Ear Service of Dr. Friesner were actively concerned. This case illustrated the importance of a diagnosis of disease involving the petrous portion of the temporal bone. X-ray examination of the petrous bone was not done. A young man, age 23 years, was admitted with a history of having had pneumonia eight months previously, followed a month later by an abscess in the region of the sternum, which proved to be an osteomyelitis. He later developed other abscesses superficial and deep in various parts of the body. A positive blood culture-staphylococcus aureus was obtained. He left the hospital apparently improved, returned some time later with a history of a right suppurative middle ear of two weeks' duration, vertigo, severe right-sided temporal headache. The subsequent course was extremely interesting, for he began to develop signs pointing to involvement of the cranial nerves in relation to the posterosuperior surface and apex of the petrous. He had intense unilateral headache, which radiated to the jaw and right corneal hypaesthesia from involvement of the Gasserian ganglion. There was a right external rectus weakness and facial paralysis. Examination of the vestibular apparatus by Dr. Friesner pointed to a nonreacting right vestibular apparatus. The auditory nerve, however, was functionating at this time. Four days later we notice that he had total loss of hearing on the right side. In view of the fact that the loss of function of the vestibular nerve preceded by a number of days the loss of cochlear function, we felt, considering the other points in the examination, that we were dealing not with a labyrinthitis but with a metastatic involvement of the petrous bone secondary to the original focus in the sternum. There was no evidence of mastoid involvement. The neurologists were inclined to the view of the possibility of an epidural abscess, and so upon their insistent request Dr. Friesner opened the mastoid—findings were entirely negative as to the mastoid, dura of the floor of middle and posterior fossa. Patient's condition became steadily worse. Temporal lobe and cerebellum were explored with negative findings.

At a limited post-mortem a deep-seated cerebellar abscess was found. Undoubtedly a more complete post-mortem would have shown an osteomyelitis of the petrous portion of the temporal bone.

Dr. Maybaum felt that the Section was to be congratulated upon having an opportunity of listening to such an interesting and instructive paper. Many of the points brought out by the reader may have important clinical value.

Dr. Friesner: I don't think Dr. Goalwin quite understood Dr. Maybaum's question. He wanted to know whether there is any similarity in structure between the mastoid and the petrous pyramid—whether if there is a marked pneumatosis of the mastoid there is likely to be a similar condition around the capsule of the internal ear. That is true from my experience in the dead house; I believe there is no question about that and, pari passue, if there is an undeveloped infantile mastoid there is likely to be a sclerosed petrosa. What Dr. Maybaum wanted, and what I want to know, is this: This man had a cancer; on two occasions I have seen a metastasis in the petrous pyramid following osteomyelitis of staphylococcus origin. The men became deaf and had meningitis sympathica. Evidently there must have been some bone lesion close to meninges responsible, because, as the result, there occurred a reaction in the cerebrospinal fluid. The process was not like those that take place from the middle ear or antrum deep petrosal cells. It was not an erosion of the labyrinthine capsule from without inward. First, the neighboring nerves were involved, etc., then the vestibular apparatus—with a very good looking ear and good hearing. The signs of the involvement of the petrous pyramid antedated the signs of disease in the middle ear. What Dr. Maybaum wanted to know was whether it is possible with this method of examination to determine the osteomyelitic process in the petrous pyramid.

Dr. Goalwin: Yes. On one occasion I saw a few foci, etc.

DR. SCHILLER: When I was in Vienna, eighteen months ago, I spent several afternoons with Professor Schüller, and he showed me a series of X-ray plates demonstrating the various canals. I asked him whether the examination of these canals by X-ray was of any clinical value and he remarked that they were. He narrated cases which he had examined and found evidence of a disease in the pyramid and solely on his findings, the otologist operated upon the patient.

DR. JOHN GUTTMAN thought that the method was of great importance from the clinical standpoint.

DR. FRIESNER: One point struck me. It seems rather bizarre to attempt to make a diagnosis of the conditions in the attic in a Roentgenogram that does not even show the ossicles. Those pictures that show projecting shelves of bone—whether that has any value from a diagnostic standpoint, seems rather doubtful.

DR. GOALWIN (closing the discussion): Of course the petrous pyramids vary in different individuals, but so do the sinuses. The right and left sphenoids are rarely alike in the same skull. The normal variation of the finer details of the petrous pyramid lies within closer limits than the variation of the sinuses. To dismiss the entire subject of the X-ray diagnosis of diseases of the petrous pyramid as a possibility, on the ground of these variations, is, to say the least, unscientific. It is the Roentgenologist's duty to determine what constitutes the normal by measurements on a large number of skulls in the skeleton and in the living. Even if we assume that one cannot tell what is normal for the size of the internal auditory meatus or the semicircular canals, or the attic, the fact still remains that a complete or partial *destruction* of either of these structures is beyond question pathological, that the attic is normally separated from the antrum by the aditus, and when the attic and antrum are merged into a single cavity we are dealing with some pathological process. The reason why different Roentgenologists obtain a superimposition of the external and internal auditory meati with different inclinations of the head to the plate is that they use a wedge which places *the lateral wall* of the skull at a definite angle. Since the inclination of this wall to the median plane of the skull varies we naturally get an *apparent* variation. Our practice is to mark the median plane on the skull as described, and measure the inclination from *that plane*. When that is done our experience corresponds with that of Schüller, who gives this inclination as 25-30.°

It makes no difference what reference point you choose as long as you *measure* the position of the head. Since Dr. Dixon believes that the petrous pyramids vary so much that one cannot make a definite diagnosis from their roentgenograms, it is rather curious that they are sufficiently regular to give him a superimposition of the two meati of the same ear with a *constant* inclination of 13.°

I am sorry Dr. Law gained the impression that I claimed to be the originator of the idea that the internal and external meati should be superimposed in mastoid Roentgenology. I stated specifically in the beginning of my paper that the lateral view was devised by Schüller, the posteroanterior axial view by de Kleyn and Stenvers, and the top view by Mayer; that the mastoid positions have been described by *Sonnenkalb, Heidenhain and Law.* The only modification which I made in all these positions is that I keep the central ray vertical and the plate horizontal, so that the manipulations are limited to the skull. I find that the additional tilting of the head which this involves can be done with comfort to the patient, and without carrying the mastoid or the pyramid any farther away from the plate than is done by the older methods. Our method furthermore enables us to use the Bucky diaphragm, which cannot be done when the tube is inclined to the plate. I believe that even for mastoid diagnosis at least two views, Schüller's and Sonnenkalb's, should be used.

Dr. Maybaum's first question takes us into the relation of the diseases of the orbit and sinuses to the petrous pyramid, which we cannot go into at the present time. Acusticus tumors do show a spindle-shaped enlargement

of the internal canal when they are situated within the canal. When situated without the canal they show a pathognomonic sign on the X-ray picture which has been described by Schüller. This consists of a thinning of the dorsum sellae and marked tipping of it forward. Later the entire dorsum may be destroyed, and still later the sella turcica is involved. *All of Dr. Schüller's cases have been proven by operation or autopsy.* One has to be very careful in making a diagnosis of acusticus tumor from enlargement of the internal meatus alone, as they do vary normally on the opposite sides of the same skull. Here as elsewhere it is not the size that counts as much as the shape. When one internal meatus is twice as large as the other, or when it shows erosion of the walls, we can safely say that we are dealing with a pathological process.

If Dr. Maybaum means by "the relation between the type of pyramidan type of mastoid" the relation in degree of pneumatization, his question can be answered in the affirmtive. A highly pneumatized pyramid usually goes with a highly pneumatized mastoid. The type of cells is, on the other hand, not always the same, as they vary even in the same mastoid process. This relationship was definitely demonstrated on the normal right side of one of the cases shown. On the left side of the same case the atrophy of the mastoid cells, combined with the marked sclerosis of the pyramid, indicates, to us, tuberculosis of the mastoid upon which was superimposed a pyogenic infection of the middle ear. We believe this to be particularly likely in view of the marked enlargement and atrophy of the walls of the attic of the supposedly normal right side.

DR. GOALWIN (answering Dr. Friesner): Yes, foci of infection, such as tuberculous caries, metastatic abscess, etc., can be demonstrated on the X-ray picture, which foci may have no connection whatever with the vestibular or auditory apparatus.

DR. FRIESNER: It seems rather bizarre that an X-ray picture which shows so clearly the attic, as Dr. Goalwin's pictures do, should not show the ossicles. Some of the pictures showed shell-like structures projecting into the attic. Are those of any value from a diagnostic standpoint? Dr. Goalwin is quite right when he says that the aditus is normally extremely narrow and that when it is sufficiently large to show on the X-ray picture it is pathological.

DR. GOALWIN: Only part of the attic shows in normal cases. It is that part which is projected over the external auditory canal in Mayer's top view of the pramid. The double shadow of attic and canal makes the former easily visible. The ossicles, on the other hand, cannot be seen, because thicker and denser bone is projected over them. The shape of a diseased attic or antrum, the regularity or irregularity of the walls, the presence of increased or decreased density, etc., all have an important value from a diagnostic standpoint. Enlargement of these cavities when associated with smooth walls of normal density is indicative of cholesteatoma. When the walls are smooth but increased in density it is indicative of pyogenic infection. When the walls are smooth but decreased in density it indicates tuberculosis. When the walls are irregular, the outline dentate, etc., it is indicative of carcinoma. When two or more of these conditions exist at the same time the diagnosis becomes more difficult, but is usually possible.

So far as I know, the technique described has not demonstrated any finer details than I have shown.

In the diagnosis of transverse fractures of the pyramid one must be careful not to be misled by the sphenotemporal fissure which is projected over the pyramid near the apex in the posteroanterior Stenvers view.